The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 141, No. 13 — CHICAGO, ILLINOIS — NOVEMBER 26, 1949

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DIAGNOSIS AND TREATMENT OF ACUTE CORONARY DISEASES

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Coronary disease is the commonest of the heart diseases,[1] and it is essential that we emphasize the importance of the distinction between acute coronary artery occlusion and acute coronary insufficiency.[2] These two diseases are completely separate entities in themselves.[3] Since acute coronary occlusion is probably an end result of long-standing arteriosclerosis and thus cannot be prevented, the treatment is merely symptomatic. But in acute coronary insufficiency the treatment is prophylactic, preventive and curative. Acute coronary insufficiency is as common as acute coronary occlusion, and therefore it is essential that the practicing physician understand it as well as he does acute coronary occlusion. Because 25 to 40 per cent of patients with coronary disease show nothing abnormal on physical examination and in the resting electrocardiogram,[4] it is of paramount importance to perform the exercise electrocardiogram on such persons before excluding coronary insufficiency.

We feel that the incidence of acute coronary insufficiency is just as common as that of acute coronary occlusion.[3d] We have had occasion to report that the incidence of coronary occlusion in this country is about one-half to one million attacks annually.[5] Naturally, if one includes the ordinary episodes of angina pectoris, the incidence of acute coronary insufficiency would be far greater than that of acute coronary occlusion. Even accepting only those cases in which severe illness or necrosis of the myocardium occurs, we feel it is just as common as acute coronary occlusion. In the experience of the medical examiner, acute coronary insufficiency with myocardial infarction is more common than acute coronary occlusion.

The simplest way to make the distinction between acute coronary occlusion and acute coronary insufficiency [2] is to think of the latter as an episode of angina pectoris, varying from the simple transitory bout of chest pain or pressure to severe pain or collapse due to prolonged or severe ischemia in which infarction occurs. Coronary occlusion, then, is the complete closure of an artery, whereas coronary insufficiency is a disproportion between the nutritional requirements of the myocardium and the actual blood supply or coronary flow.

Pathologically there is a difference.[6] In coronary occlusion the underlying disease is invariably arteriosclerotic, and subintimal hemorrhage, hematoma or thrombosis is the mechanism for the acute occlusion. The myocardial infarction of the left ventricle is usually massive, through and through from the endocardium to the pericardium and confined to the distribution of the occluded artery. The subendocardial region is usually most severely involved, but endocardium and pericardium are also injured. Since the endocardium is damaged, mural thrombosis occurs and embolization is frequent. Because the pericardium is involved, pericarditis is common. In acute coronary insufficiency, however, a different picture is present. The coronary artery may be normal, but usually there is sclerosis with narrowing. The areas of necrosis, if present at all, are usually disseminated, patchy and often microscopic. The subendocardial region may be the only region involved, particularly the papillary muscles. There is no mural thrombosis or embolization, since the endocardium is not implicated. The subendocardium of the left ventricle is most frequently the site of injury because it is most susceptible to curtailment of blood supply. The endocardium itself receives blood from the cavity of the heart. During the isometric contraction of the ventricle the intramural pressure is greatest near the subendocardium and least near the pericardial region. It has been shown experimentally that the intramyocardial pressure nearest the endocar-

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Read before the Section on General Practice at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 8, 1949.

1. White, P. D.: Heart Disease, New York, The Macmillan Company, 1944, p. 476. Master, A. M.; Jaffe, H. L., and Dack, S.: The Incidence of the Various Types of Heart Disease: A Post Mortem Study, J. Mt. Sinai Hosp. **9**: 658 (Nov.) 1942. Deaths and Death Rates for Each Cause, United States, 1944-1946: Special Reports National Summaries, Vital Statistics, Federal Security Agency, Public Health Service, National Office of Vital Statistics, (Aug. 3) 1948, vol. 29, no. 2, p. 25.

2. Master, A. M.; Gubner, R.; Dack, S., and Jaffe, H. L.: Differentiation of Acute Coronary Insufficiency with Myocardial Infarction From Coronary Occlusion, Arch. Int. Med. **67**: 647 (March) 1941. Master, A. M.; Dack, S., and Jaffe, H. L.: Nomenclature in Coronary Artery Disease, Mod. Concepts Cardiovas. Dis. **10**: no. 11 (Nov.) 1941. Master, A. M.; Jaffe, H. L.; Dack, S., and Grishman, A.: Coronary Occlusion, Coronary Insufficiency, and Angina Pectoris, Am. Heart J. **27**: 803 (June) 1944.

3. (*a*) Master, A. M.: Coronary Artery Diseases in Industry, Compens. Med. **1**: 3 (April) 1946; (*b*) Progress in Acute Coronary Diseases: Acute Coronary Artery Insufficiency With and Without Acute Occlusion, New York Med. **2**: 19 (May 5) 1946; (*c*) Acute Coronary Insufficiency: A New Concept of Acute Coronary Disease, U. S. Nav. Med. Bull. **47**: 226 (March-April) 1947; (*d*) Acute Coronary Artery Disease: History, Incidence, Differential Diagnosis and Occupational Significance, Am. J. Med. **2**: 501 (May) 1947. (*e*) Master, A. M.; Dack, S.; Grishman, A.; Field, L. E., and Horn, H.: Acute Coronary Insufficiency: An Entity: Shock, Hemorrhage and Pulmonary Embolism as Factors in Its Production, J. Mt. Sinai Hosp. **14**: 8 (May-June) 1947.

4. White, P. D.: Heart Disease, New York, The Macmillan Company, 1944, p. 827. Montgomery, G. E., Jr.; Dry, T. J., and Gage, R. P.: Further Observations on the Prognosis in Angina Pectoris Due to Coronary Sclerosis: A Study of Four Hundred and Five Patients Who Survived Ten or More Years, Minnesota Med. **30**: 162, 1947.

5. Master, A. M.; Jaffe, H. L., and Dack, S.: The Prevalence of Coronary Artery Occlusion, New York State J. Med. **39**: 1937 (Oct.) 1939. Master, A. M.: Incidence of Acute Coronary Artery Occlusion: A Discussion of the Factors Responsible for its Increase, Am. Heart J. **33**: 135 (Feb.) 1947.

6. Horn, H.; Field, L. E.; Dack, S., and Master, A. M.: Pathologic and Physiologic Aspects of Acute Coronary Insufficiency: An Analysis of Twenty-Five Cases of Subendocardial Infarction, to be published.

dium is the greatest. In other words, the subendocardial region, although ordinarily receiving the greatest blood supply, is at the same time subjected to the most wear and tear and is the most susceptible to anoxemia.

Differentiation of Acute Coronary Insufficiency from Acute Coronary Occlusion

	Acute Coronary Insufficiency	Acute Coronary Occlusion
Definition	Inadequacy of coronary circulation due to disproportion between the nutritional requirements of the myocardium and coronary blood flow	Closure of coronary artery with infarction of area supplied by the vessel
Physiologic mechanism	Decreased supply of oxygen to the myocardium or disproportion between supply and demand of latter	Complete occlusion of a coronary artery
	Transient to prolonged myocardial anoxia or ischemia	Complete ischemia
Pathology	1. Artery: variable involvement from normal to severe sclerosis	1. Artery: invariably arteriosclerotic, acute occlusion
	2. Myocardium: focal disseminated involvement of left ventricle	2. Myocardium: confluent massive infarction of left ventricle
	3. Area of damage: subendocardial; papillary muscles	3. Area of damage: through and through infaret from endocardium to pericardium, confined to distribution of the occluded artery
	4. No mural thrombosis or embolization	4. Mural thrombosis and embolization frequent
	5. No pericarditis	5. Pericarditis common
Symptoms A. Severity	Negligible to marked Varies with severity of precipitating mechanism and degree of myocardial ischemia and necrosis	
B. Pain	Transient to prolonged; variable from none to severe; usually relieved by glyceryl trinitrate	Prolonged; not relieved by glyceryl trinitrate; may aggravate; usually severe
C. Gastrointestinal	Nausea and vomiting not present in a mild attack	Nausea and vomiting common
Signs A. Fever	None or slight	Constant; 100 to 103 F. usually
B. Cardiovascular 1. Shock	Usually absent	Commonly present
2. Heart sounds	Usually no change	Poor, diminution of 1st sound, tic-tac rhythm, embryocardia, gallop
3. Pericardial rub	Absent	May be present
4. Blood pressure	Usually no change; may rise during pain, may fall in shock	Definite fall most common; rarely may rise during pain
5. Tachycardias and arrhythmias	Usually absent except when a precipitating agent	Common after the onset
6. Heart failure	None to variable	Left heart failure common
Laboratory findings	None if simple transient episode of angina pectoris	Leukocytosis and rapid sedimentation rate; vital capacity reduced
	Changes often slight; if prolonged, moderate leukocytosis and elevated sedimentation rate	Circulation time prolonged
Electrocardiographic findings	Characteristic pattern: 1. RS-T depressions	Characteristic pattern: 1. RS-T elevations progressing to deeply
	2. T wave inversions	2. Inverted T waves
	3. Q waves rare	3. Large Q waves
	4. No reciprocal relationship between leads 1 and 3	4. Reciprocal relationship between leads 1 and 3
	5. Changes usually reversible and of short duration	5. Rarely completely reversible
	6. Often diagnosed by 2-step exercise test before or after acute stage	
Etiologic factors and treatment	Prevention and prophylaxis: initiate treatment immediately and energetically to prevent physiologic state from becoming an anatomic result; abolish precipitating factor	1. Symptomatic 2. Treatment of complications 3. Heparin or "dicumarol" or both
	A. Due to increased cardiac work 1. Elimination of physical and emotional strain	
	2. Coronary vasodilators: glyceryl trinitrate, nitrites, xanthines	
	3. Paroxysmal tachycardia: digitalis, quinidine, vagopressor drugs, etc.	
	4. Hypertensive crises: vasodilators, phlebotomy, sedation	
	5. Thyrotoxicosis: propylthiouracil, iodine, radioactive iodine, surgery	
	6. Infections: penicillin, streptomycin, sulfonamide drugs	
	B. Due to diminished coronary flow 1. Shock and hemorrhage a. Restoration of circulating blood volume, blood pressure and hemoglobin b. Fluid, plasma, transfusions	
	2. Hypotensive crises a. In addisonian crisis: fluids, salt, adrenal cortex extract	
	b. Following sympathectomy and spinal anesthesia: ephedrine, "paredrine," phenylephrine hydrochloride	
	3. Prevention and treatment of hypoglycemic shock: restricted use of insulin in the presence of coronary arteriosclerosis; glucose as needed	
	4. Acute heart failure: digitalis, mercurials, ammonium chloride, salt-free diet	
	5. Spontaneous angina pectoris: coronary vasodilators	
	C. Due to anoxemia 1. Anesthesia: adequate oxygen to prevent anoxia: ether, etc. when sympathetic stimulation desired; cyclopropane, etc. when parasympathetic	
	2. Acute anemia: transfusion of whole blood or packed red cells	
	3. Acute pulmonary disease and asthma: oxygen, aminophylline, epinephrine, chemotherapy	
	4. Carbon monoxide poisoning: oxygen and blood transfusion	
	5. Pulmonary embolism: early ambulation postoperatively; heparin and "dicumarol"; papaverine, atropine; oxygen, femoral vein ligation	

The age distribution is about the same for coronary insufficiency and coronary occlusion except that the former may occur at any age, since it may be present in the absence of coronary artery sclerosis. In coronary occlusion the structural predisposing factors are coronary arteriosclerosis, diabetes, hypertensive heart disease and xanthomatosis. In coronary insufficiency the structural predisposing factors are coronary arteriosclerosis, coronary ostial stenosis, valvular heart disease (especially aortic stenosis and mitral stenosis) and cardiac hypertrophy. It is evident that in the case of "tight" mitral valves not enough blood will be ejected by the left ventricle into the aorta and into the coronary arteries and therefore coronary insufficiency ensues. The same reasoning holds in aortic stenosis. In cardiac hypertrophy, although the fiber

size increases, capillary blood supply remains unchanged. Functional predisposing factors for coronary insufficiency are congestive heart failure, chronic anemia, hyperthyroidism and hypothyroidism.

As to the direct precipitating factors in coronary occlusion,[7] we are proponents of the school which accepts no cause except, perhaps, shock and shocklike states. In coronary insufficiency, however, there are direct etiologic factors.[8] Again, if one thinks of the causes of angina pectoris, one knows the precipitating factors of coronary insufficiency. There are three main causes, and the combination of the three is probably the commonest of all. The first cause is increased cardiac work, for example, severe exertion or emotional stress, cold, excitement, eating, sexual intercourse, straining at stool, extremes of temperature, tachycardia, hypertensive or hyperthyroid crises, acute infectious states, drugs such as epinephrine and, finally, simultaneous overindulgence in food, tobacco and alcohol. The second chief cause of coronary insufficiency is diminished coronary flow. Clinically, shock, hemorrhage, operation and trauma are the most important factors, and their clinical significance cannot be overemphasized. Hypotensive crises such as occur in Addison's disease, orthostatic hypotension, spinal anesthesia, sympathectomy and acute heart failure also produce a decrease of the coronary flow. Reflex vasoconstriction from pulmonary embolism, peripheral thrombosis or embolism, acute abdominal conditions such as gastroenteritis, gallbladder disease, complicated hiatus hernia and pancreatitis are additional causes of diminution in coronary blood supply.

The third important etiologic factor of acute coronary insufficiency is impaired oxygenation of the blood such as occurs during anesthesia, high altitude, asphyxia, carbon monoxide poisoning, acute anemia, pulmonary embolism, asthmatic attacks and pulmonary insufficiency. It is insufficiently realized that morbidity and mortality in hemorrhage often are produced by ischemia or necrosis of the left ventricular myocardium. Similarly, myocardial injury in carbon monoxide poisoning may be more important than respiratory or cerebral involvement.

The symptoms in acute coronary insufficiency vary from negligible, indefinable pressure or pain in the chest to severe pain. The pain in acute coronary occlusion, on the other hand, is generally prolonged and severe, not relieved by glyceryl trinitrate, which, in fact, may aggravate it. Nausea and vomiting are characteristic classic symptoms of coronary occlusion, but they do not occur in a mild or average attack of coronary insufficiency.

The temperature in acute coronary occlusion usually reaches 100 to 103 F. Cardiovascular signs are common: There are poor heart sounds with diminution of the first sound, tic-tac rhythm, embryocardia and gallop rhythm; pericardial rub may be present; the blood pressure invariably falls and rarely rises during the pain; tachycardias and arrhythmias are frequent; left heart failure is common. In acute coronary insufficiency, however, there is little or no fever, shock is usually absent, heart sounds are rarely altered, pericardial rub is absent and there is either no change in the blood pressure or it may actually rise in the attack of angina pectoris. If shock is present, the blood pressure may fall. Tachycardias and arrhythmias are usually absent unless they themselves are the precipitating causes of coronary insufficiency. Heart failure is usually not observed.

The laboratory findings in acute coronary occlusion are leukocytosis, rapid sedimentation rate and prolongation of the circulation time, whereas in acute coronary insufficiency these are usually absent or slight. If the attack is severe, leukocytosis and elevated sedimentation rate, of course, do occur.

The electrocardiogram [8] in acute coronary occlusion is characteristic, with RS-T elevations, progressing to inverted T waves, deep Q waves and a mirror-like relationship between leads 1 and 3. These changes last for a long time and are rarely completely reversible. It is important to emphasize the specificity of the electrocardiogram in acute coronary occlusion. The only other disease in which the electrocardiogram may simulate it is dissecting aneurysm. We have seen 3 or 4 cases in which the dissection involved the coronary arteries directly or the hemorrhage in the pericardium occurred around these vessels and compressed them. On the other hand, the electrocardiogram of coronary insufficiency reveals RS-T depressions and T wave inversions but no Q waves and no reciprocal relationship between leads 1 and 3. The changes are usually reversible and of short duration.

It should be emphasized that coronary insufficiency may occur in the presence of a normal electrocardiogram. The diagnosis in such cases can be established by the Master two-step exercise test,[9] performed, of course, only after the acute stage of the illness. For example, we have elicited evidence of coronary insufficiency (precordial pain and electrocardiographic changes) in patients with acute or chronic anemia who have been given the exercise test. After treatment of the anemia, the result of the exercise test became negative, indicating amelioration of the coronary insufficiency. Even in cases of severe coronary disease with angina pectoris the resting electrocardiogram may be entirely normal and the two-step exercise test, by producing transient RS-T and T wave changes, is of great value in confirming the clinical diagnosis.

In more than 75 per cent of patients who have recovered from the acute attack of coronary occlusion, abnormalities in contraction of the left ventricle will be seen by fluoroscopy or roentgenkymography.[10] Paradoxic pulsation with systolic expansion will be observed in nearly half the patients. In acute coronary insufficiency, however, alterations in cardiac contraction are generally not observed.

The treatment of acute coronary occlusion is symptomatic and aimed to prevent complications. Heparin or dicumarol® or both are now in general use to prevent embolic complications.[11] Entirely different is the situa-

7. Master, A. M.; Dack, S., and Jaffe, H. L.: The Role of Effort, Trauma, Work and Occupation in the Onset and Subsequent Course of Coronary Artery Occlusion, M. Ann. District of Columbia, **10:** 79 (March) 1941. Master, A. M.: Effort, Trauma, Occupation and Compensation in Heart Disease, Bull. New York Acad. Med. **17:** 778 (Oct.) 1941.

8. Footnotes 2 and 3.

9. Master, A. M.; Friedman, R., and Dack, S.: The Electrocardiogram After Standard Exercise as a Functional Test of the Heart, Am. Heart J. **24:** 777 (Dec.) 1942. Master, A. M.: The Two-Step Electrocardiogram: A Test for Coronary Insufficiency, Ann. Int. Med., to be published.

10. Master, A. M.; Gubner, R.; Dack, S., and Jaffe, H. L.: The Diagnosis of Coronary Occlusion and Myocardial Infarction by Fluoroscopic Examination, Am. Heart J. **20:** 475 (Oct.) 1940. Sussman, M. L.; Dack, S., and Master, A. M.: The Roentgenkymogram in Myocardial Infarction: I. The Abnormalities of Left Ventricular Contraction, ibid. **19:** 453 (April) 1940.

11. Nichol, E. S., and Page, S. W., Jr.: Dicumarol Therapy in Acute Coronary Thrombosis: Results in Fifty Attacks With Review of Data on Embolic Complications and Immediate Mortality in Myocardial Infarction, J. Florida M. A. **32:** 365-370 (Jan.) 1946. Peters, H. R.; Guyther, J. R., and Brambel, C. E.: Dicumarol in Acute Coronary Thrombosis, J. A. M. A. **130:** 398 (Feb. 16) 1946. Wright, I. S.: Experiences with Dicumarol (3,3 Methylene-Bis-4-Hydroxycoumarin) in the Treatment of Coronary Thrombosis with Myocardial Infarction, Am. Heart J. **32:** 20 (July) 1946.

tion in coronary insufficiency, in which the treatment is preventive and curative and aimed at the removal of the factor precipitating the coronary insufficiency. Initial treatment, immediate and energetic, may prevent the physiologic state of myocardial ischemia or anoxemia from progressing to the anatomic state of necrosis or infarction.[3]

Consider acute coronary insufficiency due to increased cardiac work. Physical and emotional strain must be eliminated, and it is important to use coronary vasodilators such as glyceryl trinitrate, nitrites, xanthines and papaverine. In paroxysmal tachycardia, the acute coronary insufficiency precipitated by the pronounced tachycardia and increased cardiac work may dominate the clinical picture and is relieved only by measures to abolish the tachycardia, such as digitalis, quinidine or vagopressor drugs. We have observed that precordial pain, shock and electrocardiographic signs of myocardial ischemia disappear abruptly after cessation of the tachycardia. Similarly, in hypertensive crises, the coronary insufficiency produced by the decided increase in cardiac work has been relieved by measures to reduce the elevated blood pressure, such as vasodilators, sedation and phlebotomy, if necessary. In toxic goiter we have repeatedly observed the aggravation of coronary insufficiency due to underlying coronary sclerosis. We have seen an intractable anginal syndrome associated with electrocardiographic abnormalities disappear completely after thyroidectomy or medical treatment of the thyrotoxicosis, such as strong iodine solution U. S. P. (Lugol's solution), radioactive iodine or propylthiouracil. In acute infections, particularly pulmonary infections, the administration of such agents as sulfonamide drugs, penicillin, streptomycin and aureomycin will prevent coronary insufficiency and myocardial involvement.

In acute coronary insufficiency due to diminished coronary flow, treatment is aimed at increasing coronary flow. In spontaneous severe angina pectoris, large doses of coronary vasodilators are helpful. In shock and hemorrhage, restoration of circulating blood volume, blood pressure and hemoglobin by intravenous infusion of fluid, plasma and blood may be life saving. We have observed many patients in the coronary age group, and even younger, in whom a sudden massive gastrointestinal hemorrhage produced severe coronary insufficiency manifested by a clinical state simulating acute coronary occlusion, particularly when the internal hemorrhage was unrecognized. Although such therapy may be contraindicated in acute coronary occlusion, the administration of repeated transfusions and the control of the hemorrhage have abolished the coronary insufficiency. In hypotensive crises due to Addison's disease, intravenous use of fluids (saline) and adrenal cortex extract abolish coronary insufficiency. Similarly, after sympathectomy and spinal anesthesia and in orthostatic hypotension, the maintenance of normal blood pressure with ephedrine, paredrine hydrobromide® (p-hydroxy-*a*-methyl-phenylethylamine hydrobromide) and phenylephrine hydrochloride (neo-synephrine hydrochloride®) prevents diminished coronary flow. In patients with diabetes treated with insulin, proper regulation of the dosage to prevent hypoglycemic shock is necessary, particularly in the presence of coronary sclerosis. In acute heart failure, treatment with digitalis, mercurial diuretics and salt-free diet aids in the restoration of normal coronary blood flow by improving myocardial tonus and increasing cardiac output. Reflex coronary vasoconstriction may occur in intra-abdominal conditions such as cholecystitis. In a woman with arterial hypertension and severe angina pectoris due to coronary sclerosis, it was believed that the angina pectoris was aggravated by an associated cholelithiasis. Despite the almost intractable angina pectoris, a cholecystectomy was performed, after which there was a dramatic amelioration of the coronary insufficiency and anginal syndrome.

In coronary insufficiency due to anoxemia, restoration of normal blood oxygenation has been curative. During surgery, anoxia due to anesthesia must be prevented by adequate oxygen intake. Ether is the anesthetic of choice when sympathetic stimulation is desired and cyclopropane when parasympathetic stimulation is indicated. It is no longer acceptable to "push" the anesthesia in patients with heart disease or other predisposing factor of coronary insufficiency. Sufficient oxygen must always be available. In acute anemia the restoration of normal blood hemoglobin by transfusion of whole blood or packed red cells is necessary for the prevention or treatment of myocardial anoxia. It is not unusual for severe angina pectoris and abnormal electrocardiographic changes to appear during acute anemia and to disappear completely when the anemia is treated rapidly. Acute pulmonary insufficiency due to acute pulmonary infection, bronchial asthma and emphysema is a common precipitating cause for acute coronary insufficiency. The electrocardiographic findings may even suggest the presence of myocardial infarction. The treatment of the pulmonary insufficiency with oxygen, epinephrine, aminophylline and antibiotics often causes the coronary insufficiency and electrocardiographic changes to disappear entirely. Similarly, in acute cor pulmonale due to pulmonary embolism, death may actually be caused by infarction of the myocardium due to diminished coronary blood flow resulting from shock and myocardial anoxia. Early ambulation postoperatively, the administration of oxygen, heparin, dicumarol® and papaverine and femoral vein ligation, all measures for prophylaxis and therapy of the pulmonary embolism, are also curative for the coronary insufficiency. Finally, in carbon monoxide poisoning, death due to myocardial anoxia and infarction can be prevented by artificial respiration, administration of oxygen and replacement transfusions.

In regard to prognosis, in acute coronary occlusion a fatal outcome occurs in 5 to 25 per cent of patients.[12] The duration of the illness is weeks to months, and the condition soon after attack is usually poor. Evidence of myocardial infarction is often permanent. It is a prolonged illness, which may leave its earmarks for years. In acute coronary insufficiency the prognosis is variable but better as a whole; it depends on the precipitating factor. It is only occasionally fatal. The duration of the illness is minutes, days or weeks. The condition after an attack is good in a simple episode of angina pectoris; otherwise it is variable. The degree of recovery is complete for a single attack of angina pectoris; it is variable otherwise.

In our opinion acute coronary occlusion is not directly compensable, because it is not influenced or precipitated

12. Master, A. M.: Prognosis in Occlusion of the Coronary Arteries, Mod. Concepts Cardiovas. Dis. **5**: no. 12 (Dec.) 1936. Master, A. M.; Jaffe, H. L., and Dack, S.: The Treatment and the Immediate Prognosis of Coronary Artery Thrombosis (Two Hundred and Sixty-Seven Attacks), Am. Heart J. **12**: 549 (Nov.) 1936.

by factors in the external environment. Acute coronary insufficiency, on the other hand, may be definitely compensable.

It is clear, then, that acute coronary insufficiency, like acute coronary occlusion, is a complete entity with its own definite predisposing and precipitating factors, with its peculiar pathology and physiology, with a characteristic electrocardiogram and, most important of all, with a preventive, prophylactic and curative treatment.

ABSTRACT OF DISCUSSION

DR. B. S. OPPENHEIMER, New York: When one sees case after case in which sudden exceptionally heavy physical exertion is followed by good evidence of acute coronary occlusion, one is compelled to accept a causal relationship between the so-called accident and the coronary thrombosis. Trauma to the chest, with or without evidence of contusion or hemorrhage in the chest wall, can undoubtedly precipitate coronary occlusion. Dr. Master would probably agree that direct severe trauma to the chest may cause coronary occlusion. When it comes to the relation of severe psychic trauma, I believe that I have seen some instances in which it is difficult to deny the causal relationship of the psychic shock to the coronary thrombosis; however, such isolated examples may really be only coincidental. As to therapy, morphine is of great value in relieving the agonizing pain of an acute episode. In the angina of coronary insufficiency or the pain of coronary occlusion it is a godsend. Its effect is largely on the central nervous system, but one must acknowledge its dramatic relief of acute pulmonary edema. There is evidence of other pharmacologic effects. Papaverine is most useful for the pain of subacute and long-standing coronary disease. I have been administering it since about 1930, when Rössler published his convincing experimental evidence of its value. Papaverine is not an addictant. For those instances of refractory angina pectoris or even status anginosus, inhalation of 95 to 100 per cent oxygen several times a day, but never for over one hour at a time, has been helpful. One may give it six or more times a day. The treatment of the low blood pressure in cardiac shock with phenylephrine hydrochloride (neo-synephrine hydrochloride®) (0.5 to 1.0 cc. of a sterile 1 per cent solution given subcutaneously or intramuscularly) is of value. The use of procaine and its derivatives should be avoided; of those available, none is without danger. Caution must be observed in the use of epinephrine in the acute coronary episodes. Digitalis may be used if there is heart failure in coronary artery occlusion. If there is no heart failure I do not hesitate to prescribe quinidine for auricular fibrillation complicating coronary artery thrombosis. But in general it may properly be questioned whether it is not better to treat the pathologic-physiologic disturbances like congestive failure without being too energetic in attempting the rapid radical abolition of the arrhythmias. If the cardiac rate in ventricular tachycardia is too high, that in itself is a danger. Sound clinical judgment should underlie evaluation of the proper position of the arrhythmia in the patient's illness, that is, whether elimination of the arrhythmia is really required. Many of the arrhythmias complicating acute coronary occlusion will terminate spontaneously with watchful waiting.

DR. HYMAN I. GOLDSTEIN, Camden, N. J.: If ever a speaker deserved to have his name attached to a syndrome, certainly "Master's syndrome" should be the name for coronary insufficiency, which Master has emphasized for the members of the profession. I ask the author to comment on the "Master two-step exercise" in the study of the functional integrity of the coronary vessels. I also call attention to the fact that, in certain severe anemias and in polycythemia, there is a certain element of coronary insufficiency; in the first because of the lack of oxygen in the blood, and in the second because of the heavy blood and increased viscosity due to the polycythemic state. Hence, in some severe anemias, relief of pain follows the administration of effective liver extract, iron, and blood transfusion. The same is true in polycythemic patients who get relief of cardiac distress and pain due to coronary insufficiency when the polycythemic state is relieved by bleeding, iron deficient diet and radioactive phosphorus (P^{32}). Similar cardiac pain is observed in hyperthyroidism, and with high thyroid dosage in hypothyroid patients. Archangelo Piccolomini (1586) nearly four hundred years ago said: "I am of the opinion that sudden blockage or obstruction of the cord or vessel to the heart may often explain sudden and unexpected departure from this life" (coronary thrombosis).

DR. ARTHUR M. MASTER, New York: Dr. Oppenheimer brought up the question of compensability. A definite distinction must be made between acute coronary artery occlusion and acute coronary insufficiency in this respect. My associates and I do not believe that we know of anything that precipitates acute coronary occlusion except perhaps shock and shocklike states. If one works on a compensation board, or if one is a referee, one begins to think that everything precipitates coronary occlusion. However, if one is a clinician, and takes the histories in the hospital wards, day in and day out, on hundreds of patients as we have done, one does not obtain a story of unusual effort before the coronary occlusion. Occasionally there must be a coincidence, since a million attacks of coronary occlusion occur yearly and every one exerts unusual effort at some time. May I remind Dr. Oppenheimer that the stories that the workmen give at the compensation board hearings are frequently different from the histories they relate to the first physician they see, or in the hospital. Concerning trauma, one must again distinguish between coronary occlusion and coronary insufficiency. At autopsy one does not find an acute occlusion following trauma. In a steering wheel accident, for example, one practically always sees the picture of pericarditis or of coronary insufficiency. I agree with Dr. Oppenheimer that morphine is invaluable in treatment of acute coronary episodes, but I believe that the pendulum has swung too far again. When physicians give too much morphine the patient becomes constipated and distended, with detriment to his cardiovascular function. At least 40 per cent of patients with coronary disease do not present any objective evidence of heart disease. The physical examination, chest roentgenogram and resting electrocardiogram all may appear to be entirely normal. When the resting electrocardiogram is normal, the two-step exercise test is of decided value. In other words, under a little physiologic stress changes will appear in the electrocardiogram. This is logical, for the patient with angina pectoris experiences chest pressure on effort, and when he rests his pain is relieved. I agree with Dr. Goldstein that anemia may produce coronary insufficiency and that treatment of the anemia will benefit the patient.

Bone Growth.—Stephen Hales in 1727 scientifically recorded his observations on bone growth and thus initiated a study which has since grown in scope and magnitude. Using a half-grown chick, he pierced two holes in its legs at a measured distance with a sharp-pointed iron and found that two months later the marks were still equidistant, although the leg had grown 1 inch (2.54 cm.) in length. He concluded that this growth in length was from the epiphysis and that proper nutritive matter was necessary for its growth. Duhamel confirmed this in 1743 in the pigeon and in the dog. The experiments of John Hunter showed more accurately that normal longitudinal growth of the shaft of a long bone occurred only at the epiphysis. Also, by using Belchier's observation that animals fed on madder root had a red tinge to their bones, he showed that the increase in diameter of bone was due to new bone being formed on the exterior of the shaft. These conclusions have been confirmed by Compere, Müsenbach, Humphry, Wegner, Haas, Gatewood and Mullen, Payton, Selye, Phemister, Bisgard and Bisgard, Siegling and Aries. From these studies it can be said positively that normal longitudinal growth of the shaft occurs only at the epiphysis. That this growth is from the diaphysial side of the epiphysis was further shown by Brucke, who rotated the epiphysial cartilages in rabbits 180 degrees, demonstrating that bony apposition took place only on the side originally facing the diaphysis.—Sterling G. Parker, M.D., Decatur, Ill., *Archives of Surgery*, November 1949.

ROLE OF VASODILATOR DRUGS IN CORONARY OCCLUSION

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When a branch of a coronary artery is occluded, ischemia of the heart muscle follows in the area supplied by the occluded vessel. Except in the occasional case in which there has been some gradual previous inadequacy of this vessel, resulting in an increased collateral circulation to the area supplied by it, an infarction of the heart muscle results. This is the usual event. If the patient survives the initial episode, some degree of revascularization occurs, and the infarcted area again becomes a functioning portion of the myocardium. In many cases an electrocardiogram does not show evidence of the prevous infarction.

That anastomoses occur between the branches of coronary arteries was shown conclusively by Gross [1] and has been adequately confirmed by others. The subject has been lately reviewed by Blumgart.[2] Lowe [3] discussed the dynamic equilibrium that prevails between the vascular fields and showed that "tying off one branch diverted a gelatin solution into other channels which more or less completely inject the fields of the occluded vessel." The usual twenty-four hour mortality rate following experimental occlusion of the anterior descending coronary vessels of dogs is 50 per cent. Harris and Kokernot [4] have recently shown that if the occlusion is accomplished gradually over a period of approximately thirty minutes, thereby allowing the anastomotic vessels to dilate, death does not occur.

When one considers that the heart muscle deprived of its blood supply is not at rest but still has energy requirements greater than those of any other muscle, one has a higher regard for the anastomoses which do exist and one wonders that more cells do not die and that more damage is not done.

Certain factors help to reestablish equilibrium between the normal and the infarcted area. One is time to permit the smaller anastomotic arterioles to develop into more efficient blood carriers. This interval demands as complete rest for the heart as is possible. Another factor is spasm of the vessels distal to the occlusion, with a reflex vasoconstriction of the vessels in the uninvolved area. A third factor is systemic blood pressure. The coronary flow is a direct function of the mean blood pressure, and a fall of blood pressure will reduce the coronary flow and consequently the amount of blood flowing into the anastomosing vessels. Finally, and of constant importance, any agent that would tend to dilate the coronary vessels and increase the amount of blood flowing through these vessels would, it can be assumed, increase the amount of blood flowing through the collateral and anastomosing vessels. The conclusion seems inevitable that a prompt increase of the blood supply of the infarcted area would decrease the amount of damage done to the myocardial tissue and facilitate the repair of the damage already done.

Several agents have been described as increasing the coronary flow. It is essential that such agents do not lower the systemic blood pressure, with consequent decrease of the coronary flow. In most cases the blood pressure has already been lowered to a critical level. The agent used should not increase work of the heart. We are not altogether sure that the increased consumption of oxygen observed by us and others during the administration of xanthine means that there is an increase in heart work which is not compensated for by the increased coronary flow which is consequent to its use. It seems to us safe to assume that the increase of capillary bed would naturally result in a decrease of oxygen in the blood returned to the coronary sinus.

The agent should not have a peripheral vasoconstrictor effect. The decreased peripheral resistance consequent to infarction is a factor of safety. An agent which has a direct vasodilator effect on the vessel wall is preferable, one whose action does not have neurogenic effects on the tissues of other areas.

During the last few years many reports have appeared concerning the anticoagulant drugs heparin and dicumarol® as used in coronary occlusion. They have been reviewed in the report of the Committee of the American Heart Association, headed by Wright.[5] The value of heparin and dicumarol® has been conclusively established. They decrease the mortality rate in coronary occlusion, and they decrease the influence of peripheral vascular accidents. Probably these excellent results are due more to their coronary vasodilator effect and less to their anticoagulant effect. Thrombosis is not a simple matter of coagulability; it involves a number of variables and not merely the prothrombin time.

As pointed out previously,[6] when the coronary occlusion reaches the attention of the physican, it has already occurred and an infarct is already present. In many cases it has been present several hours or days. Whatever part the coagulability of the blood has played in this occurrence is done and over. It does not seem reasonable that any anticoagulant drug could effect a change in what is already an accomplished fact. There is no evidence that the thrombosis of the coronary vessel increases. Foord [7] showed at autopsy that the thrombus is only a matter of several millimeters, and the evidence is against propagation. In a certain proportion of the cases coronary occlusion is not due to primary thrombosis of the vessel but to a subintimal hemorrhage which, of itself, may occlude the vessel, or on which thrombosis may be superimposed. Wartman [8] carefully examined 41 hearts at autopsies on patients who died of acute myocardial infarction and found significant subintimal hemorrhage of the coronary vessels in 20 (49 per cent). Horn and Finkelstein [9] conducted a similar study on 172 hearts, and their findings were in accordance with those of Wartman;

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Read before the Section on Internal Medicine at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 8, 1949.

1. Gross, L.: The Blood Supply to the Heart in Its Anatomical and Clinical Aspects, New York, Paul B. Hoeber, 1921.

2. Blumgart, H. L.: Coronary Arteries as End Arteries, Am. J. Med. **5**: 321-323 (Sept.) 1948.

3. Lowe, T. E.: Some Principles Governing the Supply of Blood to the Myocardium in Occlusive Arterial Disease, Am. Heart J. **21**: 326-344 (March) 1941.

4. Harris, A. S., and Kokernot, R. H.: Late Development of Ectopic Ventricular Activity Following Coronary Occlusion, Federation Proc. **8**: 68, 1949.

5. Wright, I. S.: The Use of Anticoagulants in the Treatment of Diseases of the Heart and Blood Vessels, Ann. Int. Med. **30**: 80-91 (Jan.) 1949.

6. Gilbert, N. C., and Nalefski, L. A.: The Effect of the Anticoagulant Drugs upon the Coronary Flow, J. Lab. & Clin. Med. **34**: 797-805 (June) 1949.

7. Foord, A. G.: Embolism and Thrombosis in Coronary Disease, J. A. M. A. **138**: 1009-1012 (Dec. 4) 1948; Personal communication to the authors.

8. Wartman, W. B.: Blood, Heart and Circulation, Washington, D. C., The Science Press, 1940; Occlusion of the Coronary Arteries by Hemorrhage into Their Walls, Am. Heart J. **15**: 459-470, 1938.

9. Horn, H., and Finkelstein, L. E.: Arteriosclerosis of the Coronary Arteries and the Mechanism of Their Occlusion, Am. Heart J. **19**: 655, 1940.

significant subintimal hemorrhage was present in 100 (59 per cent) of the hearts studied. Paterson,[10] as did Nelson,[11] reported similar investigations. They found subintimal hemorrhage present in 89 per cent and 65 per cent, respectively, of the hearts examined. Wartman[12] repeated his work on an additional 77 hearts in 1948-1949 and this time found the incidence of subintimal hemorrhage of the coronary vessels to be 58 per cent. Indeed, according to Doles,[13] a hemorrhagic tendency inducing such subintimal hemorrhage is the determining factor in coronary occlusion. He advocated administration of vitamin K preparations. The mortality rates (17.2 per cent for the total series, 9.4 per cent for untreated patients, 4.7 for inadequately treated ones and 3.6 per cent for adequately treated ones) are comparable with the mortality rates of other reports.

The coronary vasodilator effect of these anticoagulant drugs might also be deduced from the reports of beneficial effect in other forms of heart disease, such as angina pectoris and cardiac insufficiency, in which there is no reason to expect any result from anticoagulant action.[14]

In a series of experiments on animals reported elsewhere,[6] it was possible to show that both dicumarol® and some forms of heparin have a direct effect on the coronary vessels in dilating them and increasing the volume of the coronary flow. This was shown to be a direct effect on the vessel wall by experiments on the empty beating heart. In the case of heparin, the effect was slight or moderate and occurred only when heparin sodium N. N. R. was used. Dicumarol® had a constant effect, equal to that of theobromine and sodium acetate U. S. P. and, we have reason to think, more prolonged. For the past several years we have routinely used one or another of the purine base derivatives in our cases of coronary occlusion because of their coronary vasodilator effect.

Experimental studies of these drugs appeared soon after Askanazy[15] reported the clinical benefits observed in angina pectoris. Hedbom[16] and Loeb[17] showed experimentally that the coronary vasodilator action was a direct effect on the vessel walls. The early results have been confirmed by many investigators in experimental work on excised arteries and on the flow in the empty beating heart, and by using a Morawitz cannula in the coronary sinus, or a Rein thermostromuhr on the coronary artery of the anesthetized animal, with the heart in situ. An increase in flow has also been shown in conscious animals, walking about with a thermostromuhr on the coronary artery. There have been abundant reports of the clinical value of these drugs, as well as some dissident reports. Our personal experience has convinced us that these drugs are of great value in coronary disease in general. In coronary occlusion the results obtained with them closely parallel the results obtained with the anticoagulants.

Barnum, Gilbert and Fenn[18] recently reported on the results of using these agents in a series of 118 cases in St. Luke's Hospital, Chicago. These were unselected, consecutive cases, in all of which the same simple treatment was given and nothing else. Atropine, 0.6 mg. (1/100 grain), and aminophylline, 0.5 Gm. (7½ grains), were administered at once. Pantopon® (a mixture of hydrochlorides of opium alkaloids), morphine or meperidine hydrochloride N. N. R. (demerol hydrochloride®) were given for pain at the onset. Oxygen was started at once, on the patient's arrival, in all cases. Atropine was used only at the onset, and phenobarbital U. S. P. and belladonna leaf U. S. P. were given routinely during the hospital stay. As soon as possible, aminophylline injection U. S. P. was discontinued and theobromine calcium salicylate or the alkaloid theobromine were substituted. No other drug was used, and there were no other variables. The mortality rate was 12.7 per cent. Thromboembolic complications for this series were 5.9 per cent. In only 2 cases of the entire series were primary venous thrombi of the legs incurred. We consider that the decrease in thromboembolic phenomena was due to the increase of the myocardial blood supply effected by the coronary vasodilator drugs. As a result of the increased blood supply, there was less damage of the endocardium and hence there was less tendency toward the formation of mural thrombi. These results are quite consonant with the results following the use of the anticoagulant drugs in cases reported elsewhere by others.

We do not question the value of anticoagulant therapy in coronary thrombosis; we do question the mechanism of its action. We also question the propriety of its being used outside a hospital equipped to follow daily the anticoagulant effects. Even under such conditions it is not entirely free from danger.

SUMMARY

A therapeutic agent which increases the volume of the coronary blood flow by direct vasodilator action offers, in our opinion, the best means for decreasing the damage that may be done to the myocardium by coronary occlusion. Experimental evidence indicates that the anticoagulant drugs heparin and dicumarol® have such a direct coronary vasodilator effect.

Clinically the results obtained with heparin and dicumarol® parallel those with coronary vasodilator drugs of the xanthine series.

The conclusion seems inevitable that increasing the blood supply to the infarcted area would decrease further damage of the myocardial tissue and facilitate repair of the damage already done.

ABSTRACT OF DISCUSSION

DR. ROY W. SCOTT, Cleveland: The authors in this paper attempt to prove that the beneficial results obtained by the use of dicumarol® and heparin in treating coronary occlusion with myocardial infarction are not due to the anticoagulant action of these drugs but rather to their action as coronary vasodilators. In support of this proposition, there are presented two types of evidence, experimental and clinical. As clinical evidence the authors refer to the results obtained in one hospital in 108 patients treated by more or less conventional therapy but with no dicumarol®; they point out that the over-all mortality rate

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14. Nichol, S. E., and Fassett, D. W.: An Attempt to Forestall Acute Coronary Thrombosis, South. M. J. **40**: 631-637 (Aug.) 1947. Foley, W. T., and Wright, I. S.: Long Term Anticoagulant Therapy for Cardiovascular Diseases, Am. J. M. Sc. **217**: 136-142 (Feb.) 1949. Coogan, T. J.: Personal communication to the authors.

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17. Loeb, O.: Ueber die Beeinflussung des Koronarkreislaufs durch einige Gifte, Arch. f. exper. Path. u. Pharmakol. **51**: 64-83, 1904.

18. Barnum, D. R.; Gilbert, N. C., and Fenn, G. K.: The Use of Xanthines in the Treatment of Acute Coronary Thrombosis, to be published.

of 12.7 per cent and the incidence of thromboembolic complications, 5.9 per cent, compare favorably with those reported by others who used anticoagulants. The largest and only well controlled study of anticoagulant therapy in the treatment of coronary occlusion with infarction was that recently reported by the American Heart Association's committee, whose chairman was Dr. Irving Wright. This comprised 432 patients who received anticoagulant therapy in addition to conventional therapy and 386 patients in the control group who received only conventional therapy. In the dicumarol®-treated series the mortality was 15 per cent and in the control group, 24 per cent. Thromboembolic complications occurred in 25 per cent of the controls and in 11 per cent of the treated patients. Evidently the mortality rate and the incidence of thromboembolic complications were both lower in the authors' 108 uncontrolled cases than in the dicumarol®-treated patients mentioned. But such data are not comparable and hence have no statistical significance. To use them as evidence that dicumarol® dilates the coronary bed in man seems to be an attempt to prove what one wants to believe. The hypothesis merits further study. Some evidence of the effectiveness of the drug as a coronary dilator in man might be obtained from its use in patients with coronary insufficiency and the anginal syndrome. I would emphasize, as the authors have, the danger of dicumarol® therapy. There probably have been many more fatalities from hemorrhage than have been reported in the literature. Valuable as the drug is in the treatment of coronary occlusion, it should never be given unless the facilities for adequate laboratory and clinical controls are available.

Dr. W. M. Fowler, Iowa City: There is a tendency in the practice of medicine to overemphasize the newer methods of therapy and in the first wave of enthusiasm to allow the pendulum to swing too far. Dr. Wright and his committee for the American Heart Association have shown beyond doubt that the use of dicumarol® lowers the mortality rate and reduces thromboembolic complications which follow myocardial infarct. The statistics which they have presented are impressive. Dr. Gilbert has suggested that the action of the drug may be more than that of an anticoagulant and that it may have some vasodilating action as well. This is an important observation. I do not believe that anticoagulant therapy should replace entirely the established methods of treatment for acute coronary occlusion. It has been shown experimentally in the dog that the size of the myocardial infarction following ligation of the coronary artery is definitely smaller in those animals treated with vasodilator drugs than in the untreated control animals. I believe that the use of such drugs in combination with oxygen therapy reduces the extent of myocardial injury in man following coronary occlusion and thereby reduces the mortality rate, the extent of the mural thrombus and the thromboembolic complications. I believe that these methods of therapy should not be discarded but should be used in conjunction with anticoagulant therapy. When given under supervision and with adequate laboratory control, dicumarol® is a valuable therapeutic agent. However, it is used indiscriminately with little laboratory control in ambulatory patients with coronary disease, it is given to patients with cardiac failure but without myocardial infarction, and I have seen it given to a patient with cardiac disease simply because a rather high thrombocyte count was observed. Such uncontrolled administration of the drug is dangerous. When properly administered, it is a valuable therapeutic aid, regardless of its mode of action.

Dr. L. A. Nalefski, Chicago: With respect to the question of statistical analysis raised by Dr. Scott, we are aware that the method of sampling we employed is not in accordance with the standard procedure. We were merely interested in seeing the mortality rates in our patients following thromboembolic phenomena. Our impression was that our results with the xanthine treatment were considerably lower than those usually reported, and this we found to be true when the 118 consecutive cases were analyzed.

DISEASE RESEMBLING NONPARALYTIC POLIOMYELITIS ASSOCIATED WITH A VIRUS PATHOGENIC FOR INFANT MICE

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ERNEST W. SHAW, M.D.
and
JOSEPH L. MELNICK, Ph.D.
New Haven, Conn.

During the summer of 1948, an unusual number of patients with illnesses resembling nonparalytic poliomyelitis were admitted to hospitals in the southern part of New England. Similar cases had also been noted in the previous year in this area as well as elsewhere.[1] Evidence had been obtained that the illness in some of the patients was caused by strains of poliomyelitis virus.[2]

It seemed possible, however, that not all the patients had poliomyelitis but that some of the mild, nonparalytic illnesses might have been caused by a different infectious agent. In order to explore this possibility, information was sought concerning the distribution of paralytic and nonparalytic cases in the southern part of New England during 1948, and certain representative patients were studied to determine whether poliomyelitis virus or some other agent could be shown to be causative of their illnesses.

As reported elsewhere, strains of a filtrable virus, resembling that first described by Dalldorf and his associates,[3] were recovered from feces of patients with nonparalytic illnesses in New England as well as from feces, sewage and flies collected during 1948 in widely separated areas of the United States.[4] This agent produced a fatal systemic infection with paralysis and myositis in suckling mice and hamsters [5] but not in older mice or hamsters or in rhesus monkeys. These observations [6] indicated the existence of an infectious agent which had not been recognized previously and the close association of this agent with illnesses resembling poliomyelitis. Further investigations seemed warranted and were made of the agent itself and of the illnesses with which it appeared to be associated.

National Research Council fellow in medical sciences (Dr. Shaw).

This study was aided by a grant from the National Foundation for Infantile Paralysis, Inc.

From the Section of Preventive Medicine and Department of Pediatrics, Yale University School of Medicine.

We are indebted to Dr. James C. Hart, director of the Bureau of Preventable Diseases, State of Connecticut Department of Health, for data on the incidence of poliomyelitis in Connecticut and to Dr. Raymond F. Mc Ateer, chief, Division of Communicable Diseases, State of Rhode Island Department of Health, for similar data from Rhode Island.

The following physicians aided in obtaining specimens and information for this study: Drs. Edward West and Alexander A. Jaworski, Charles V. Chapin Hospital, Providence, R. I.; Dr. F .M. Burke, John James McCook Memorial Hospital, Hartford, Conn., and Dr. Richard O'B. Shea, Englewood Hospital, Bridgeport, Conn.

1. (*a*) Brochner-Mortensen, K., and Raaschou, F.: Poliomyelitis anterior acuta paralytica og meningitis lymfocytaria, Ugesk. f. laeger **45**: 1263, 1948. (*b*) Silverthorne, N.; Goodfellow, A. M.; Anglin, C.; Rhodes, A. J.; Roy, T., and Snelling, C. E.: Non-Paralytic Poliomyelitis, Canad. M. A. J. **60**: 356-359, 1949. (*c*) Sabin, A. B., and Steigman, A. J.: Poliomyelitis Virus of Low Virulence in Patients with Epidemic of "Summer Grippe or Sore Throat," Am. J. Hyg. **49**: 176-193, 1949.

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3. (*a*) Dalldorf, G., and Sickles, G. M.: An Unidentified, Filtrable Agent Isolated from the Feces of Children with Paralysis, Science **108**: 61-62, 1948. (*b*) Dalldorf, G.; Sickles, G. M.; Plager, H., and Gifford, R.: A Virus Recovered from the Feces of "Poliomyelitis" Patients Pathogenic for Suckling Mice, J. Exper. Med. **89**: 567-581, 1949.

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5. Dr. Gilbert Dalldorf has suggested that the term "Coxsackie group of viruses" be used as a provisional designation for those viruses which induce severe destructive lesions of striated muscles, with or without encephalomalacia, in immature mice and hamsters. Coxsackie, N. Y., is the village where instances of human infection with this type of virus were first recognized.[3]

6. Dalldorf and Sickles.[3a] Dalldorf, Sickles, Plager and Gifford.[3b] Melnick, Shaw and Curnen.[4]

In this paper epidemiologic data will be presented to suggest the coincidental occurrence of poliomyelitis and another disease resembling it during the summer of 1948 in Connecticut and Rhode Island, and to supplement an earlier report [4] on the isolation of a new virus from some of the patients studied during this period. In describing the clinical observations it will be shown that illness associated with this agent may assume different forms resembling not only nonparalytic poliomyelitis but also epidemic pleurodynia or a mild undifferentiated febrile episode. It will be apparent that infection with this agent may represent a separate and previously unidentified disease entity.

PATIENTS AND MATERIALS STUDIED

Patients.—During 1948 in Connecticut 121 persons were reported to the state health department as having had poliomyelitis. There were no deaths. Omitting 1 patient because his illness began in 1947 and 5 patients because of insufficient data, 115 of the 121 persons reported in Connecticut as having had poliomyelitis were included in the present study. In addition, 18 patients who were not reported as instances of poliomyelitis but who were hospitalized in New Haven or Hartford with a diagnosis of aseptic meningitis, cause unknown, were included. During 1948 in Rhode Island 8 cases of poliomyelitis with no deaths were reported. One patient whose illness began in Virginia was excluded from this study. Seventeen other patients hospitalized at Providence, R. I., with the diagnosis of aseptic meningitis, cause unknown, were included.

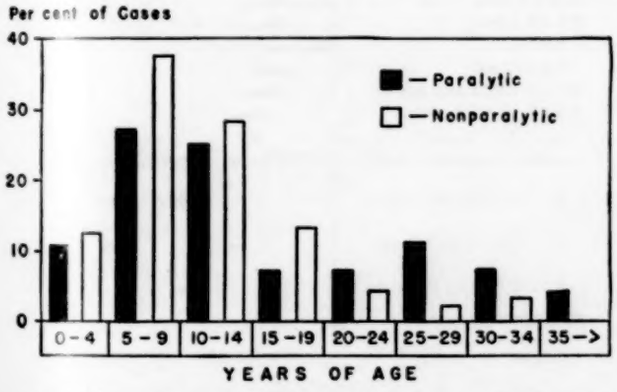

Fig. 1.—Poliomyelitis and aseptic meningitis, cause unknown, 1948, Connecticut and Rhode Island. Distribution of 44 paralytic and 113 nonparalytic cases according to age.

The patients who were studied came from homes situated in urban, suburban or rural areas in one of the two states. All but 6 of the Connecticut patients in the series were admitted to hospitals in Bridgeport, New Haven or Hartford, and all but 1 of the Rhode Island patients were hospitalized in Providence. We saw many of these patients during the acute stage of their illness; data concerning others were obtained from the hospital and health department records. All the hospitalized patients had pleocytosis of the cerebrospinal fluid. Cases of aseptic meningitis secondary to or associated with other diseases were not included in this study.

The classification of each case as paralytic or nonparalytic was necessarily arbitrary. All patients with bulbar involvement or definite localized muscular weakness were considered as paralytic. The classification of cases in which apparent paralytic involvement was mild, transitory or equivocal was usually based on the opinion of the attending physician. When opinions differed, classification was weighted in favor of the paralytic category. The 35 cases with a diagnosis of aseptic meningitis which were included in this study were clinically indistinguishable from the 78 similar cases without paralysis diagnosed as poliomyelitis. Consequently, in this paper they were all considered together as nonparalytic cases.

Materials.—Materials were collected for study from representative patients hospitalized during August, September and October, 1948, in Bridgeport, New Haven or Hartford, Conn., or in Providence, R. I. Specimens of feces collected during the acute stage of illness and following recovery were stored at— 70 C. or —20 C. Ultimately suspensions of feces were prepared and tested in newborn and older mice as well as in monkeys for the presence of viruses.[4]

Blood was also drawn during the acute and convalescent stages of illness. The serum was stored usually in a frozen state until used in various tests. Specimens of serum and feces collected from normal subjects and patients with other diseases were stored and studied in a similar manner.

TABLE 1.—*Poliomyelitis and Aseptic Meningitis, Cause Unknown, 1948, Connecticut and Rhode Island*

	Cases					
	Paralytic		Nonparalytic		Total	
State	Number	Per Cent	Number	Per Cent	Number	Per Cent
Connecticut.....	39	29	94	71	133	100
Rhode Island....	5	21	19	79	24	100
Total.....	44	28	113	72	157	100

EPIDEMIOLOGY

A total of 157 patients with a diagnosis of poliomyelitis or aseptic meningitis, cause unknown, was included in this study. As shown in table 1, 133 were from Connecticut and 24 from Rhode Island. The ratio of paralytic to nonparalytic cases in the two states was approximately the same. Forty-four (28 per cent) of the cases were classified as paralytic and 113 (72 per cent) as nonparalytic.

The geographic distribution of paralytic and nonparalytic cases within the two states was similar as determined from the location of the homes of patients in the present series who were admitted to hospitals.

A preponderance of male patients was noted in this study. As shown in table 2, 71 per cent of the patients were male and 29 per cent female. The ratio of male to female patients was almost 2 to 1 in the paralytic group and nearly 3 to 1 in the nonparalytic group.

The distribution of paralytic and nonparalytic cases in both sexes according to age was similar. As shown in figure 1, the peak incidence of cases with and without paralysis occurred in the age range of 5 to 9 years. Although both types of illness were seen in patients up to 34 years of age, there was a somewhat greater concentration of patients without paralysis in the younger age groups and a higher incidence of patients with paralysis in the older groups. Twenty-four (21 per cent) of the patients without paralysis were older than 14 years of age as compared with 16 (36 per cent) of the paralytic patients. Only 9 (8 per cent) of the nonparalytic patients were over 19 years of age as compared with 13 (30 per cent) of the paralytic patients.

TABLE 2.—*Poliomyelitis and Aseptic Meningitis, Cause Unknown, 1948, Connecticut and Rhode Island: Distribution of Cases According to Sex*

	Male		Female		Total	
Type	Number	Per Cent	Number	Per Cent	Number	Per Cent
Paralytic.....	28	64	16	36	44	100
Nonparalytic....	84	74	29	26	113	100
Total.....	112	71	45	29	157	100

In Connecticut and Rhode Island poliomyelitis has generally been most prevalent in the late summer and

early autumn. The seasonal distribution of reported cases is indicated in table 3. It will be seen that in both states over a period of many years the total number of cases in any one month was greatest in September and only slightly less in August. The peak incidence of reported cases per month during each year also occurred

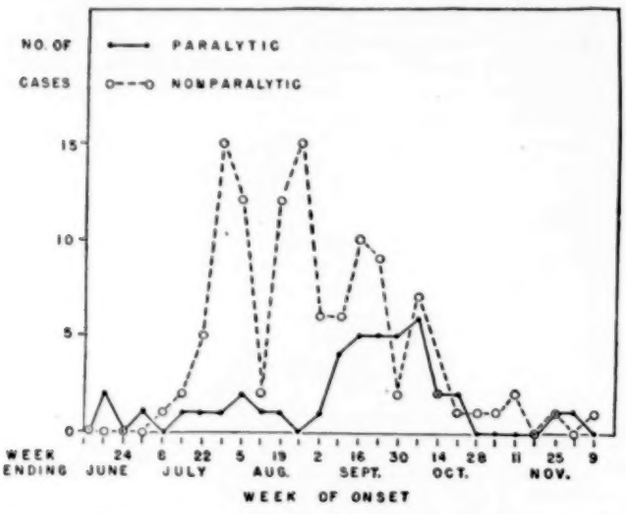

Fig. 2.—Poliomyelitis and aseptic meningitis, cause unknown, 1948, Connecticut and Rhode Island. Distribution of paralytic and nonparalytic cases according to date of onset.

most frequently in September, followed by August and October. In 1948 it occurred in August.

The distribution of paralytic and nonparalytic cases in the present series according to the date of onset is

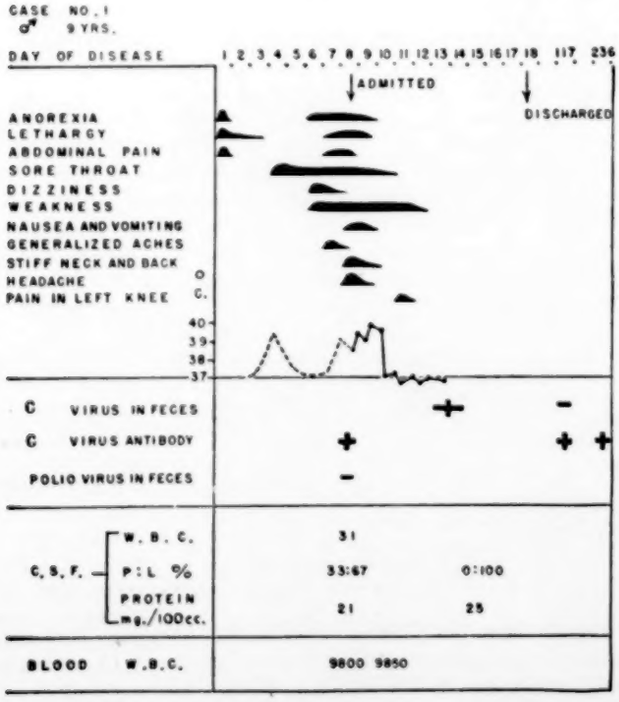

Fig. 3 (case 1).—Clinical chart and results of tests for presence of virus and antibodies in patient with syndrome of aseptic meningitis or nonparalytic poliomyelitis from whom C virus was recovered. In this and succeeding figures P: L % indicates the ratio of polymorphonuclear leukocytes to mononuclear cells, expressed as a percentage.

shown in figure 2. All but 2 cases, both paralytic, which occurred in January and March are represented.

Only 1 or 2 scattered paralytic cases per week developed during June, July and August. The weekly incidence of paralytic cases increased during September, was maximal in the first week of October and declined sharply thereafter. Although the number of these cases was not great, their distribution, with the peak of incidence per month in September, corresponded to that most commonly observed for poliomyelitis in previous years in Connecticut and Rhode Island.

The incidence of nonparalytic cases was distinctly different. Beginning in the middle of July the number of cases increased sharply to a peak at the end of July, declined in the next two weeks and then rose again to a similar peak in the fourth week of August. Thereafter, the weekly number of nonparalytic cases fluctuated but corresponded more closely to that of the paralytic cases.

A total of 37 of the 44 paralytic cases and 109 of the 113 nonparalytic cases had their onset during the sixteen weeks from July 2 to November 4. From figure 2 it can be seen that the maximal incidence of nonparalytic

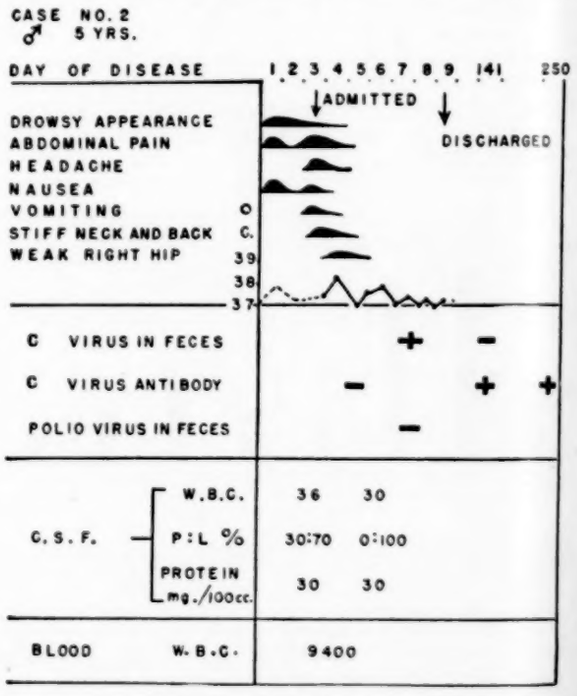

Fig. 4 (case 2).—Clinical chart and results of tests for presence of virus and antibodies in patient with syndrome of aseptic meningitis or nonparalytic poliomyelitis from whom C virus was recovered.

cases occurred in the first half of this period and of paralytic cases in the second half. During the nine weeks from July 2 to September 2 there were 8 paralytic cases as compared with 70 nonparalytic cases. But from September 3 to November 4 there were 29 paralytic and 39 nonparalytic cases. It is obvious from these data that the paralytic and nonparalytic cases were distributed differently; in fact, the difference is statistically highly significant ($P = < 0.00006$).

This observation indicated either that cases involving paralytic and nonparalytic forms of infection with poliomyelitis virus had different seasonal distribution curves, or that not all the cases included in the study had been caused by the same agent. The second alternative appeared to be substantiated by evidence obtained in the laboratory.[4]

STUDIES ON ETIOLOGY

An attempt was made to determine the cause of illness in 14 of the patients without paralysis included in the present study. Suspensions of feces obtained

from 13 of these patients during the acute illness or early in convalescence were tested in monkeys for the presence of poliomyelitis virus and were also injected into infant mice within the first few days of life.[4] The latter procedure was prompted by the report of Dalldorf and Sickles [3a] on the isolation of a virus which induced paralysis and myositis in suckling mice and hamsters from "acute phase" feces of 2 children with paralytic illnesses in the summer of 1947. Data concerning the 14 patients included in this study, together with the results of tests with their feces and serum which have been reported elsewhere,[4] are summarized in table 4. Charts to show the clinical course and laboratory findings in cases 1, 2 and 3 are included (figs. 3, 4 and 5).

Strains of a filtrable virus which caused fatal paralysis in infant mice but did not produce apparent disease in monkeys or older mice were recovered from feces obtained during the first two weeks of illness from 5 (cases 1 to 5) of the 14 patients. The virus was not found in later specimens of feces from 4 of these patients, but was found again in a specimen obtained from 1 patient (case 3) on the seventy-third day from the onset of illness. Specimens of serum obtained from these and from 5 additional patients (cases 6 to 10) during their illness or following recovery neutralized a strain (Connecticut 5) of the new virus, which had been isolated from 1 of the patients (case 2). The capacity to neutralize the virus was demonstrable in the earliest serum tested from 9 of these patients and persisted for as long as eight months, the longest interval tested. In 1 patient (case 2) the virus was not neutralized by serum obtained on the fifth day but was neutralized by serum obtained one hundred forty-one and two hundred fifty days from onset. This patient had no other illness in the interval between the first and second specimens. Suspensions of feces from 9 of these patients (cases 1 to 9) were tested in monkeys and failed to produce clinical or microscopic evidence of poliomyelitis. The illnesses in these 10 patients have been considered as representative of a disease associated with a virus pathogenic for infant mice (Coxsackie group).[5] For convenience this agent is referred to in this report as C virus.

TABLE 3.—*Summary of Data on Reported Monthly Incidence of Poliomyelitis in Connecticut and Rhode Island*

Poliomyelitis	Jan.	Feb.	Mar.	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Total Number
Connecticut—1910 to 1948 *													
Cases per month; total for 39 years.....	43	35	28	34	27	63	433	1,630	1,767	899	295	99	5,353
Month of peak incidence per year; number of years observed.....	..	..	..	..	..	..	...	11	20	7	1	..	39
Rhode Island—1916 to 1948 *													
Cases per month; total for 31 years †...	9	8	6	5	8	26	148	489	558	241	58	26	1,592
Month of peak incidence per year; number of years observed †.....	..	..	..	..	..	..	1	8	19	2	1	..	31

* Years for which monthly figures are available.
† Not including 1927 and 1934. Monthly figures not available for these years.

TABLE 4.—*Summary of Data on 14 Patients with the Clinical Syndrome of Nonparalytic Poliomyelitis or Aseptic Meningitis*

Case	Sex	Age, Yr.	Address	Clinical Diagnosis *	Onset Date, 1948	Cerebrospinal Fluid †				Duration of Illness		Results of Attempts to Isolate Viruses from Feces			Neutralization of C Virus by Patient's Serum	
						Day	White Blood Cells	P:M, Percentage	Protein, Mg. per 100 Cc.	Days of Fever	Days in Hospital	Day	Poliomyelitis Virus (in Monkeys)	C Virus (in Infant Mice)	Day	Result
1	M	9	New Haven, Conn.	N.P.P.	9/13	8	31	33:67	21	7	10	13-14	—	+	8	+‡
						15	1	.....	25			117		—	117	+
															236	+
2	M	5	New Haven, Conn.	A.M.	8/21	3	36	30:70	31	3	6	7-8	—	+	5	—
						6	30	0:100	30			141		—	141	+
															250	+
3	M	5	New Haven, Conn.	N.P.P.	9/28	5	27	7:93	19	6	12	6-10	—	+	5	+
						8	18	38:62	70			73		+	73	+
						16	2	.....	44						222	+
4	M	11	Hartford, Conn.	N.P.P.	8/27	3	125	54:46	Elevated	4	13	4	—	+	134	+
												135		—		
5	M	32	Rockville, Conn.	N.P.P.	8/23	4	92	32:68	..	9	13	4	—	+	139	+
						8	230	17:83	24			139		—		
6	M	12	Providence, R. I.	A.M.	9/13	7	600	15:85	90	6	6	1	—	—	26	+
															117	+
7	M	6	Hamden, Conn.	N.P.P.	9/21	2	129	25:75	52	3	18	5-10	—	—	2	+
						15	12	25:75	64			117		—	117	+
															229	+
8	M	18	Hartford, Conn.	N.P.P.	8/14	10	140	74:26	30	10	14	16	—	—	148	+
												148		—		
9	M	6	New Haven, Conn.	A.M.	8/22	2	42	47:53	34	1	5	6	—	—	24	+
															266	+
10	F	12	Providence, R. I.	A.M.	10/2	7	90	0:100	34	7	14	Not tested			9	+
						13	49	0:100	40						72	+
11	M	13	Monroe, Conn.	A.M.	9/3	3	46	63:37	38	4	6	6	+	—	8	—
						8	25	32:68	23			128		—	26	—
12	M	12	Stamford, Conn.	N.P.P.	8/19	2	130	44:56	60	3	18	8-9	+	—	256	—
						6	41	2:98	..							
13	M	14	Providence, R. I.	A.M.	9/11	4	142	49:51	45	2	14	6	—	—	63	—
						11	86	26:74	30			120		—	121	—
14	M	15	Terryville, Conn.	N.P.P.	8/23	2	95	.....	25	2	18	4	—	—	139	—

* N.P.P. indicates nonparalytic poliomyelitis; A.M., aseptic meningitis.
† Day indicates the day that the specimen was obtained; the date of onset is considered day 1. P:M indicates the ratio of polymorphonuclear leukocytes to mononuclear cells expressed as a percentage.
‡ Neutralization index of 1,000 or more.

Suspensions of feces from each of 2 other patients (cases 11 and 12) produced poliomyelitis in monkeys and failed to cause disease in infant mice. Specimens of serum obtained from each of these patients during or following their illnesses failed to neutralize the Connecticut 5 strain of C virus. In these 2 cases the laboratory observations supported a diagnosis of nonparalytic poliomyelitis.

Suspensions of feces from the 2 other patients (cases 13 and 14) failed to produce disease in either monkeys or infant mice, and specimens of serum obtained from each of these patients following recovery also failed to neutralize the Connecticut 5 strain of C virus. The nature of the causative agent in these 2 cases was not established; the possibility of infection with poliomyelitis virus was, of course, not excluded.

The C virus was not recovered during the period of this study from the feces of 6 patients with paralysis

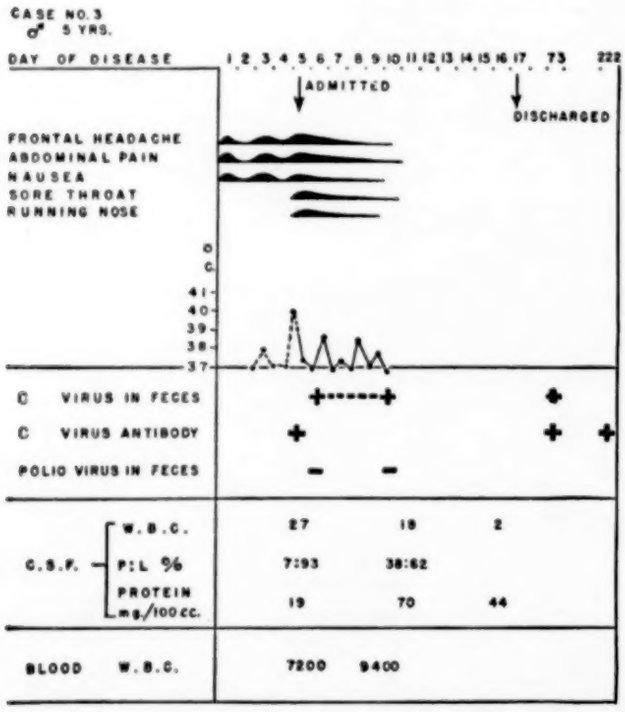

Fig. 5 (case 3).—Clinical chart and results of tests for presence of virus and antibodies in patient with syndrome of aseptic meningitis or nonparalytic poliomyelitis from whom C virus was recovered.

or from the feces of 31 other persons, including healthy subjects and patients hospitalized for various illnesses other than poliomyelitis or aseptic meningitis.

The results of tests carried out with serum from each of the 14 representative patients yielded no evidence to indicate that the virus of either mumps or Newcastle disease was responsible for the illnesses in this group.

The C virus was recovered from patients with other clinical forms of illness. A strain was found in the feces and the capacity to neutralize the virus was demonstrated in serum obtained from a boy aged 14 who was hospitalized in September 1948 for a brief illness resembling epidemic pleurodynia (fig. 6, case 15). Three persons apparently contracted infection with C virus while working with it in the laboratory.[7] The virus was recovered from their feces and in 2 cases from pharyngeal swabs or washings. In all 3 instances serum collected early in the course of illness or prior to it failed to neutralize the virus (Connecticut 5 strain), but in subsequent specimens the capacity to neutralize this strain appeared and increased in titer.

Virus pathogenic for infant mice was also found in the feces of individual patients who had nonparalytic illnesses during the summer of 1948 in Ohio. The capacity to neutralize strains of the C virus has been demonstrated in convalescent serum from some of these patients and from patients who had mild illnesses during the summer of 1947 in Baltimore. As reported previously,[4] strains of C virus have been found in pooled human feces collected during 1947 in Akron, Ohio, and in 1948 in Winston-Salem, N. C., as well as in samples of sewage collected in 1948 from six cities, three in Connecticut and three in North Carolina. Strains of virus pathogenic for infant mice have also been recovered from flies collected during the summer of 1948 in Connecticut, North Carolina and Texas. Dalldorf and his associates[3b] isolated strains of virus with similar properties from the feces of patients with apparent poliomyelitis collected in New York state and in Wilmington, Del., during 1947 and in New York during 1948.

These observations indicate that strains of virus pathogenic for infant mice are widely distributed in nature. Evidence has also been obtained that they exist in multiple serologic types.[8] Additional data will be presented to show that illnesses associated with this virus have occurred in a number of different clinical forms.

CLINICAL FEATURES

Syndrome of Aseptic Meningitis or Nonparalytic Poliomyelitis.—The clinical features of illness in 10 patients with the syndrome of aseptic meningitis or nonparalytic poliomyelitis from whom the C virus was isolated or whose serum neutralized it have been analyzed. A summary of data concerning them is presented in table 4 (cases 1 to 10), and charts illustrating the course of illness in several are shown in figures 3, 4 and 5 (cases 1, 2 and 3). These patients ranged in age from 5 to 32 years; by chance 9 were male and only 1 was female.

Symptoms: The onset of illness was gradual in 7 of the patients and relatively abrupt in 3. Prodromal constitutional symptoms generally preceded those suggestive of meningeal irritation. In 3 patients the prodromal symptoms were interrupted and the course of illness appeared to be diphasic. In table 5 the symptoms noted in these patients have been listed in order of frequency.

In each of the patients fever developed, in all but 1 on the first day of illness. The elevation of temperature persisted or recurred for a period of one to ten days, coinciding with the acute stage of illness. Headache, nausea and abdominal pain were noted frequently and occurred on the first day of illness in more than half of the patients with these complaints. Stiffness of the neck or back, and vomiting, which were also common symptoms, were present at the onset of illness in only one third of the patients with these complaints and usually did not appear until after fever and other symptoms had been present for one or more days. Half of the patients complained at some time of pain in one or more extremities. This was usually ill defined and not severe; in all instances it was of brief duration. Four patients had pain as well as stiffness of the neck or back. Unusual drowsiness or lethargy was present in 4 patients, and a like number complained of sore

7. Shaw, E. W.; Melnick, J. L., and Curnen, E. C.: Infection of Laboratory Workers with a Virus Pathogenic for Infant Mice, to be published.

8. Dalldorf, Sickles, Plager and Gifford.[3b] Melnick, Shaw and Curnen.[4]

throat. Sleeplessness, in 1 instance associated with backache, and generalized weakness were each troublesome to 3 patients. As indicated in table 5, other symptoms, including chills and manifestations of coryza, were encountered infrequently. It seemed doubtful that diarrhea, which was experienced by 1 patient for a day one week before the onset of other symptoms, was related to the illness under consideration. The occurrence of abdominal pain in 6 patients and of pain in the chest in 1 (case 7) is noteworthy in view of the emphasis on these complaints by other patients from whom the C virus was recovered. These other patients are described later in the paper.

Physical Signs: The signs of illness found on physical examination have been summarized in table 6. In general they were few and not distinctive. Most patients did not appear particularly ill or uncomfortable. All were rational and cooperative. The temperature on admission ranged from 37.6 to 38.6 C. with a mean of 38.5 C. (99.7 to 101.5 F. with a mean of 101.3 F.). The maximal temperature observed in each patient during the course of illness varied from 38.2 to 40.3 C. with a mean of 39.4 C. (100.8 to 104.5 F. with a mean of 102.9 F.). Fever lasted from one to ten days, with a mean of 5.6 days. During the acute stage of illness, the pulse rate was usually accelerated to correspond with the temperature. The respiratory rates were normal.

Stiffness of the neck or back was present in all but 1 patient at the time of admission to the hospital and was the factor in most instances which led to hospitalization. The term is used here to indicate some degree of resistance to active or passive forward flexion. Quantitative evaluation of this sign was difficult, as the observations were variously described not only from day to day but on the same day by different observers. In 5 patients the stiffness was mild or equivocal and lasted for only two or three days. In 3 patients the stiffness was mild or somewhat more pronounced and lingered for approximately ten days. In 2 patients (cases 4 and 7) persistent stiffness of the back in the first instance and of the back and neck in the other gradually diminished; these finally disappeared only after periods of one month and six weeks, respectively. In 7 patients with stiffness of the neck or back, the Brudzinski or the Kernig sign, or both, were reported to be positive.

Hyperemia of the pharynx was noted in 7 patients. This was invariably mild and of short duration. Three patients (cases 7, 8 and 10) appeared to have spasm of the hamstring muscles. A watery nasal discharge was noted in 2 patients. One patient (case 2) was thought by an observer to have slight weakness of the right leg for two days. In view of the fact that 6 of these patients complained of abdominal pain, it seems noteworthy that none showed definite signs of abdominal tenderness.

Cerebrospinal Fluid: Lumbar puncture was performed on all these patients at least once, in 6 patients twice and in 1 patient three times. Initial lumbar punctures were done on the second to eighth day and repeated on the sixth to sixteenth day of illness. No abnormalities of pressure were noted, and cultures of the cerebrospinal fluid were sterile. The observations on examination of the cerebrospinal fluid are shown in table 4. The total leukocyte counts on the initial observation ranged from 27 to 600; in all but 1 case the counts were less than 150. The leukocyte counts had decreased on the repeat examination with 1 exception. In this instance (case 5) the count which was 92 on the fourth day rose to 230 on the eighth day. The percentage of polymorphonuclear cells in the initial specimens of spinal fluid was less than 25 in 3 cases, 25 to 50 in 5 cases and more than 50 in 2 cases; the range was 0 to 74 per cent. The protein content of the spinal fluid was found to be greater than normal at some time during the course of illness in 5 of the 10 patients; in 3 patients it was observed to increase and was still elevated on the eleventh to sixteenth day. The highest level noted was 90 mg. per hundred cubic centimeters on the seventh day (case 6).

Other Laboratory Results: The total leukocyte count in the blood of these patients was usually normal; the range was between 6,900 and 9,900 with 1 exception. In this patient (case 9) a count of 21,500 recorded on the second day of illness was found to be 9,600 on the following day without any obvious explanation for the unusual leukocytosis. The differential cell counts and other hematologic observations were within normal range. Urinalyses were normal. The reaction to intradermal injection of old tuberculin in 1 to 1,000 dilution in 1 instance or purified protein derivative of tuberculin, first and second strength, was negative in the 6 younger patients tested for tuberculin sensitivity.

TABLE 5.—*Symptoms in 10 Patients with the Clinical Syndrome of Nonparalytic Poliomyelitis or Aseptic Meningitis from Whom the C Virus Was Recovered or Whose Serum Neutralized It*

Fever	10	Sleeplessness	3
Headache	9	Weakness	3
Stiff neck or back	9	Generalized aches	2
Nausea	8	Dizziness	2
Vomiting	6	Running nose	2
Abdominal pain	6	Cough	1
Pain in extremities	5	Chills	1
Pain in neck or back	4	Earache	1
Drowsiness or lethargy	4	Pain in back and chest	1
Sore throat	4	Diarrhea (one week before onset)	1

TABLE 6.—*Physical Signs of Illness in 10 Patients with the Clinical Syndrome of Nonparalytic Poliomyelitis or Aseptic Meningitis from Whom the C Virus Was Recovered or Whose Serum Neutralized It*

Signs			Number of Cases
Elevated temperature			10
	Range	Mean	
On admission	37.6 to 39.6 C.	38.5 C.	
Maximal	38.2 to 40.3 C.	39.4 C.	
Duration	1 to 10 days	5.6 days	
Stiffness of the neck or back			9
Positive Brudzinski or Kernig sign			7
Hyperemia of pharynx			7
Spasm of hamstring muscles			3
Nasal discharge			2
Weakness of extremity			1

Clinical Course: The course of illness in each of these patients was relatively brief and uncomplicated, and terminated in complete recovery. All but 2 patients (cases 4 and 7) with persisting spasm of the neck and back had fully recovered at the time of discharge. As noted in table 4, the period of hospitalization in these patients ranged from five to eighteen days with a mean of 11.3 days.

Other Forms of Illness.—In other patients from whose feces strains of the new virus were recovered, as in case 3, abdominal or thoracic pain were the most prominent symptoms. An adolescent boy, who was hospitalized during September 1948 in New Haven

because of pain in the abdomen and chest, had an illness which was considered to be epidemic pleurodynia (figure 6, case 15).

CASE 15.—L. C., a white boy aged 14, was apparently well until Sept. 24, 1948, when dyspnea and pain developed in the right side of his chest and abdomen. He felt better the next day, but on September 26 the pain returned and radiated to the right shoulder. He also complained of frontal headache and vomited several times.

On admission to the hospital he had moderate distress from the thoracic pain, which was accentuated by deep breathing. His temperature was 39 C. (102.2 F.), pulse rate 96 and respiratory rate 42. The neck was slightly resistant to anterior flexion. The thorax expanded equally on the two sides; the lungs were clear by physical and roentgenologic examination. There was questionable tenderness in the right lower quadrant on abdominal palpation. The Kernig sign was negative; the

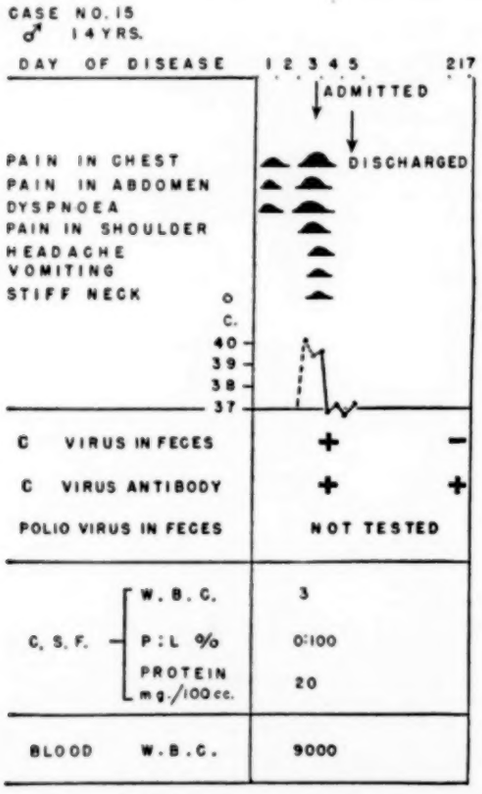

Fig. 6 (case 15).—Clinical chart and results of tests for presence of C virus and antibodies in patient with illness resembling epidemic pleurodynia.

reflexes were normal. The leukocyte count was 9,000 with 74 per cent polymorphonuclear cells. Lumbar puncture yielded normal cerebrospinal fluid.

On the day after admission of the patient the fever subsided, and the symptoms and signs of illness disappeared. He was discharged from the hospital September 28.

Acute febrile illnesses developed in 3 laboratory workers while they were studying the C virus. Details of the clinical course and laboratory observations in these cases have been reported elsewhere.[7] Their illnesses varied in severity but bore a resemblance to that in case 15, previously described. Abdominal and thoracic pain were the chief symptoms, and there were no signs to suggest involvement of the meninges or the central nervous system. All 3 patients recovered completely. These cases appeared to represent instances of infection by the C virus, presumably contracted in the laboratory.

COMMENT

The frequent occurrence during the summers of 1947 and 1948 of cases of mild illnesses resembling nonparalytic poliomyelitis suggested the possibility that some of the illnesses might have been caused by an infectious agent other than poliomyelitis virus. The investigations reported in this paper were carried out to determine whether this possibility could be shown to have validity.

Although poliomyelitis was considered to be epidemic during 1948 in the United States this was not true in Connecticut or Rhode Island, where the total number of patients reported was not unusual and none died. The 157 patients in the present study included most of those treated for poliomyelitis in this area. All had pleocytosis of the cerebrospinal fluid and clinical findings consistent with poliomyelitis. One hundred and thirteen (72 per cent) of the cases were classified as nonparalytic. They included 78 patients with a diagnosis of poliomyelitis and 35 who had similar illnesses which were called "aseptic meningitis, cause unknown" thereby avoiding the more sinister and specific term, "poliomyelitis."

An analysis based on the date of onset of each patient's illness revealed that the highest incidence of cases without paralysis was in August and of paralytic cases in September and October. In a similar study made in Denmark, Brochner-Mortensen and Raaschou [1a] compared cases of paralytic poliomyelitis and those without paralysis classified as primary lymphocytic meningitis which occurred in Copenhagen from June 1, 1947 to January 31, 1948. They found that cases of each type were distributed in regular epidemic curves with the peaks for the nonparalytic and paralytic cases in August and October, respectively. Their epidemiologic observations resemble those reported in this paper and provide a similar basis for interpretation.

In the present study the discrepancy in the weekly distribution of paralytic and nonparalytic cases, although not great in point of time, appeared none the less to be statistically highly significant. Particularly in view of this observation it seemed improbable that all the cases included in this group represented infection with a single agent, i. e., poliomyelitis virus. The alternative view that the group was heterogeneous with respect to etiologic agent and included cases in which illnesses were caused by more than one infectious agent seemed a more likely possibility, and one that could be investigated in the laboratory.

As described in the foregoing portion of the paper, studies made on 14 representative patients without paralysis provided evidence that the nonparalytic group was heterogeneous. Only 2 of these patients were found to harbor poliomyelitis virus. However, a virus pathogenic for infant mice, similar to that first described by Dalldorf and Sickles,[3a] was isolated from material from 5 of these patients. Moreover, serum obtained from 10 of the 14 patients during or following their acute illness neutralized a strain of the new virus which was recovered from 1 of them. The 10 patients whose serum neutralized the new virus included the 5 from whom this agent was isolated but not the 2 who harbored poliomyelitis virus.

Although the capacity of serum from these patients to neutralize a Connecticut strain of the new virus is not conclusive proof that their illnesses were caused by this agent, it is evidence in support of this view,

especially in those patients who were also found to have the virus in their feces during the acute stage of illness.

Further evidence that the virus is, in fact, pathogenic for human beings was obtained from a study of the illnesses in 3 workers in this laboratory, who presumably contracted infection with the agent while engaged in studying it.[7] During the acute stage the virus was recovered from feces of all 3 of these persons and from pharyngeal secretions of 2 of them. In all 3 the serologic capacity to neutralize the virus was not present prior to illness but appeared early and increased to high titer during convalescence.

The immunologic relations of this virus have not been completely established. However, it was found that homologous hyperimmune serum prepared in animals completely neutralized this virus, whereas it was not neutralized by hyperimmune serum prepared in animals against the following viruses: poliomyelitis (Lansing, North Carolina 1948 and Texas 1948 strains) Theiler's FA and GD VII strains of mouse encephalomyelitis virus, mumps, herpes simplex, lymphocytic choriomeningitis, encephalomyocarditis, louping ill, Venezuelan equine encephalomyelitis and Newcastle disease viruses.[4] Dalldorf and his associates[5b] also found that the heterologous immune serum which they tested failed to neutralize their strains of virus. Evidence is accumulating that the virus exists in multiple serologic types.[8]

Fragmentary as present knowledge is concerning the properties and distribution of this new virus, certain resemblances to poliomyelitis virus have been noted. Like poliomyelitis virus, the new virus is excreted in human feces[9] and may be recovered from pharyngeal secretions,[10] it has been recovered from flies and has been found in sewage.[4] It has been obtained from one or more of these sources in widely scattered areas. From the limited observations recorded to date it would appear that poliomyelitis and infection with the new virus may have a similar seasonal distribution. Moreover, certain clinical forms of infection by these two agents may resemble or be indistinguishable from one another.

Although in the present study the new virus and the serologic capacity to neutralize it were identified with nonparalytic patients exclusively, these patients were selected for particular study and the evidence is insufficient to assume that none of the paralytic cases represented infections with the new virus. Attempts to recover the virus from 6 paralyzed patients of the present series gave negative results. It will be recalled, however, that a similar virus was first isolated by Dalldorf and Sickles from 2 patients with paralysis.[5a] It remains to be determined how frequently and with what severity paralysis may occur in patients infected by this agent.

It seems noteworthy that 1 patient from whom this virus was recovered and whose serum neutralized it had an acute illness with the clinical features of epidemic pleurodynia. In all 3 of the workers with laboratory infections abdominal or thoracic pain were also prominent symptoms. These observations add to the variations in the clinical features of disease associated with this virus and suggest that further search be made for a similar agent in patients with illnesses resembling not only poliomyelitis but also pleurodynia or epidemic myalgia.

9. Footnotes 5 and 6.
10. Footnotes 4 and 7.

From present knowledge it appears that strains of a virus pathogenic for infant mice are widely distributed in nature. This agent has also been found to be pathogenic for human beings, and acute illnesses associated with it may resemble poliomyelitis, epidemic pleurodynia or a mild, undifferentiated fever. The possibility of inapparent infection with this agent has been indicated in man[7] and proved in chimpanzees.[4]

SUMMARY

A study was made of 157 patients with a diagnosis of poliomyelitis or aseptic meningitis, cause unknown, who were admitted during 1948 to hospitals in Connecticut and Rhode Island. All of the patients had pleocytosis of the cerebrospinal fluid; none died. Forty-four (28 per cent) of the cases were classified as paralytic and 113 (72 per cent) as nonparalytic.

An analysis based on the date of onset of each patient's illness showed that the highest incidence of nonparalytic cases was in August and of paralytic cases in September and October. The difference in distribution was found to be statistically highly significant. Together with evidence obtained in the laboratory, this appeared to indicate that not all of the patients included in the study had diseases caused by poliomyelitis virus.

Suspensions of feces from 13 representative patients without paralysis were injected into mice and monkeys. Strains of a virus which differed from poliomyelitis virus were recovered from 5 of these patients. The virus was pathogenic for infant mice but not for older mice or rhesus monkeys. A Connecticut strain of this virus was neutralized by serum obtained during or following illness from 10 of the nonparalytic patients, including the 5 from whom the agent was isolated. Poliomyelitis virus was recovered from 2 of the nonparalytic patients whose feces did not yield the new virus and whose serum did not neutralize it.

During the period of this study, the new virus was sought but not found in feces from 6 patients with paralysis and in feces from 31 other persons including healthy subjects and patients hospitalized for various illnesses other than poliomyelitis or aseptic meningitis.

A strain of virus pathogenic for infant mice was recovered from feces collected from a boy in September 1948, during an acute illness which resembled epidemic pleurodynia. Serum obtained from him during and following the illness neutralized this virus. Abdominal or thoracic pain were also prominent symptoms of the acute illnesses in 3 laboratory workers who contracted infections with this virus while studying it.

An analysis was made of the clinical features in 10 patients with the syndrome of aseptic meningitis or nonparalytic poliomyelitis from whom the new virus was isolated or whose serum neutralized it. The observations were similar to those in cases attributed to infection with poliomyelitis virus.

From present knowledge it appears that strains of a virus pathogenic for infant mice are widely distributed in nature and that in man acute illnesses associated with this agent may resemble poliomyelitis, epidemic pleurodynia or a mild undifferentiated fever.

The observations in this study suggest that a common disease caused by this virus may exist. Its occurrence during the summer of 1948 in the southern part of New England may account for the discrepancy noted in the distribution of paralytic and nonparalytic cases and for the mild character of many of the illnesses which were considered to be poliomyelitis.

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ASEPTIC MENINGITIS OF NEW VIRUS ORIGIN

A Series of Eighteen Cases

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This series of 18 cases of aseptic meningitis observed at the Charles V. Chapin Hospital from July through November 1948 is noteworthy because at no time in the past were patients with similar cases of aseptic meningitis ever admitted to the hospital. By method of exclusion, relying heavily on extensive laboratory virus studies performed at the Yale University Medical School Virus Laboratory, New Haven, Conn. (Drs. Paul, Curnen and Shaw [1]), and at the National Institutes of Health Virus Laboratory, Bethesda, Md. (Dr. K. Habel [2]), the etiologic agent in this series of cases is shown to be a new virus affecting the meninges in a mild and typical manner. A new virus disease entity with characteristics similar to those observed in this series has been recently noted by Webb and associates [3] and by Paul, Curnen and Shaw.[1]

CLINICAL DATA

The nature of our cases was most characteristic, with strikingly close similarities in the histories, examinations and hospital course of the patients. The first patient was admitted July 21, 1948; 7 and 8 patients were admitted during August and September, respectively, and 1 each in October and November. The peak of incidence occurred about September 1. Twelve patients were from Providence and the other 6 were from scattered areas in Rhode Island.

Histories of the present illness were similar with few variations. The protean manifestations were typical of meningeal irritation: headache, moderate aching and stiffness of the neck, and occasional vomiting. All patients experienced the generalized effects of any acute illness, namely: fever, minimal anorexia and general malaise. The onset varied from two to four days before admission with initial symptoms of slight upper respiratory infection and minimal throat irritation in half of the patients and fever and malaise in the others. In the usual history, the patient was bedridden for about two days prior to hospital entry, with increased malaise and persistent, severe, frontal and occipital headache unrelieved by acetylsalicylic acid. These symptoms increased in severity, the headache descending to the posterior aspect of the neck and causing minimal pain on anterior flexion of the head.

There were no exposures to measles, chickenpox, mumps, herpes and, most important, anterior poliomyelitis. Twelve of these patients (two thirds of the series) had already been admitted before the first patient with paralytic anterior poliomyelitis for 1948 was hospitalized.

All were well developed and well nourished persons, the average age being 14 years. Only 3 of the 18 were females. On admission they were alert, cooperative and apprehensive; they complained chiefly of severe diffuse headache and moderate general malaise. Positive observations were limited to fever, 101 to 103 F., moderate redness of the oral pharynx and stiffness of the neck. Cervical stiffness can be described further as definitely not nuchal rigidity, but 1 plus stiffness and pain in the posterior aspect of the neck elicited only by forced anterior flexion of the head. Neurologic examinations revealed essentially normal conditions. One third of the patients had a positive Kernig's sign.

Nose and throat cultures were negative; results of urinalyses, blood counts and cell differential counts were all normal. Results of spinal fluid examinations, however, were all abnormal and consistent with a typical virus type of inflammatory reaction of the meninges. The spinal fluids were clear with elevated cell counts, lymphocytes predominating, averaging 154 per cubic millimeter. Spinal fluid protein was increased in each case and averaged 65 mg. per hundred cubic centimeters (normal up to 30 mg.). Sugar and chloride levels and manometric pressures were normal, as were stained smears and cultures of the spinal fluid.

There was a rapid recovery and uneventful convalescence in each patient. Within twenty-four to forty-eight hours there was complete disappearance of the intense headache and a remarkable transformation from acutely ill, apprehensive patients to cheerful asymptomatic persons anxious for discharge. The only physical abnormality to persist more than two days was the neck stiffness and neck pain, which lasted as long as six days but did not cause incapacitation to the patient and could only be elicited by forceful anterior flexion of the head. Treatment was limited to bed rest and symptomatic therapy. Fluid obtained by a repeat lumbar puncture on the seventh to tenth hospital day still showed slightly increased protein and cell count values in most cases, usually one half of the initial values.

The average hospital stay was thirteen days. Although two weeks of hospitalization was considered advisable, no ill effects were noted in 2 male patients discharged against advice on their fifth and sixth hospital days. Thirteen patients of the group were interviewed three months after the acute illness and found to be in excellent physical and mental health. A summary of the cases is given in the accompanying table.

VIRUS STUDIES

The etiologic agent of these 18 cases of aseptic meningitis was discovered by a process of laboratory exclusion of the common viruses. Virus studies, contrary to the commonly held opinion that they are meager and limited, are relatively plentiful and readily available for many specific virus diseases.[4] Virus entities considered were nonparalytic poliomyelitis, lymphocytic choriomeningitis, mumps encephalitis, St. Louis type of American epidemic encephalitis, herpes encephalitis, and encephalitis associated with exanthematous diseases.

Because these cases occurred at the height of the expected poliomyelitis season in a year of increased incidence of poliomyelitis in the United States,[5] the diagnosis of abortive or nonparalytic poliomyelitis was strongly suspected. Yet only 6 cases of paralytic poliomyelitis were diagnosed in Rhode Island in 1948 compared with 81 and 82 in the previous two years. Since it is possible to detect the poliomyelitis virus from vari-

Drs. Paul, Shaw, Melnick, Habel and especially Curnen gave helpful assistance in this study.

From the Department of Pediatrics, Charles V. Chapin Hospital, Dr. Harold G. Calder, chief of pediatric service.

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3. Webb, C. H.; Wolfe, S. G., and Simpson, V.: Clinical Features of a Summer Disease (Three Day Fever), Apparently of Virus Etiology, New Orleans Med. and Surg. J. **101**: 249-256 (Dec.) 1948. Webb, C. H.; Wolfe, S. G., and Howitt, B. F.: Three Day Fever, an Epidemic Febrile Disease of Childhood, Presented at the Annual Session of American Medical Association, Atlantic City, N. J., June 8, 1949.
4. Smadel, J. F.: The Practitioner and the Virus Diagnostic Laboratory, J. A. M. A. **136**: 1079-1081 (April 24) 1948.
5. Health Officer's Report, Public Health Service, Washington, D. C., Jan. 1, 1949.

ous sources, most easily from stools,[6] less likely from the oral pharynx [7] and, rarely, from the blood,[8] stool specimens were obtained from 10 patients during the first two days of their acute febrile illness and submitted to the Yale virus laboratory. Specimens were reported negative for the poliomyelitis virus after being processed and inoculated intracerebrally into rhesus monkeys (Macaca mulatta).[1] This ruled out the poliomyelitis virus, as positive results after inoculations into monkeys are most frequent in active cases of poliomyelitis of either the paralytic or nonparalytic type.[9]

The clinical course of these persons was distinctly different from that seen in poliomyelitis patients. The brief illness was due to meningeal irritation of moderate severity. Headache and not cervical spasm was the main observation. Recovery was more rapid, the average age was higher and the spinal fluid contained more lymphocytic cells per cubic millimeter and a higher protein level than in the average case of poliomyelitis, paralytic or nonparalytic.

desired criteria were kept in mind with speculation and the hope of isolating a weak and mild strain of poliomyelitis virus which could be used for immunization.

Last year, as was indicated previously, exactly similar cases of aseptic meningitis were observed independently for the first time by Paul, Shaw and Curnen [1] and were tentatively considered a new virus entity. Recently Melnick, Shaw and Curnen [10a] have described the isolation of a new virus from the feces of patients whose disease was diagnosed as nonparalytic poliomyelitis or aseptic meningitis during the summer and autumn of 1948 in four cities in southern New England, which included Providence and 4 of our series of 18 cases. The virus isolated was found to be pathogenic only to newborn suckling albino mice, producing in them weakness and paralysis accompanied with diffuse myositis. They [10a] were also able to demonstrate neutralizing antibodies in the convalescent serums of the majority of the patients studied. In regard to the 4 patients from our series, Melnick and associates did

Summary of 18 Cases of Aseptic Meningitis

Case	Age, Yr.	Fever	Stiff Neck	Headache	Malaise	Spinal Fluid: Cells, per Cu. Mm.	Spinal Fluid: Protein, Mg./100 Cc.	White Blood Cells	Hospital Days
1	19	++	+	+++	0	100	60	9,000	9
2	12	++	+	+++	++	370	50	5,000	12
3	7	++	+	++	+++	275	70	10,000	5
4	15	++	+	+++	++	120	75	8,500	7
5	5	++	0	0	0	105	50	12,000	16
6	12	++	+	+++	0	100	55	11,500	13
7	19	++	+	+++	++	18	50	7,000	12
8	32	++	+	+++	+++	196	60	5,000	14
9	31	++	++	+++	+	85	35	7,500	14
10	19	++	+	+++	+++	200	75	7,000	17
11	12	++	+	0	0	600	90	7,000	6
12	14	++	+	+++	++	75	45	6,500	25
13	13	++	+	0	0	160	125	11,000	24
14	13	++	+	++	+	200	90	8,000	27
15	7	++	+	++	0	70	70	9,000	13
16	16	++	+	+++	+++	21	45	9,000	16
17	11	++	+	++	++	91	55	7,000	15
18	17	++	+	+++	++	100	65	9,000	10

Had the poliomyelitis virus been identified and isolated, further tests would have been performed on convalescent serums being stored at the Yale Virus Library to determine the antibody titer by monkey neutralization tests and to establish the fact that the virus possessed adequate antigenic immunologic antibody stimulation. The two components of a strain of virus causing mild symptoms and yet producing immunity by sufficient antibody response are the desired criteria of the long sought poliomyelitis virus vaccine. Immunization with a poliomyelitis virus vaccine was unsuccessfully attempted by Leake [10] in 1935. Although the virus in our series of cases was not isolated, the not report the isolation of any virus, but serum neutralizing antibody tests for their new virus were positive in 2 patients. Webb and associates [3] recently reported a new virus disease entity, three day fever, characterized by fever, headache, malaise and occasional myalgia without spinal fluid changes, and negative virus studies except in 2 cases, in which a virus identified and isolated in newborn mice and hamsters caused a decided myositis in the skeletal musculature on histologic section. In 1948 Dalldorf and Sickles [11] were the first to demonstrate from the feces of 2 paralytic poliomyelitis patients a new virus pathogenic only to newborn suckling mice, producing in them paralysis with diffuse myositis.

Clinically, lymphocytic choriomeningitis was a distinct possibility, as this disease is also characterized by a brief meningeal course and a favorable recovery.[12] It can be diagnosed only by serum or virus laboratory

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11. Dalldorf, G., and Sickles, G. M.: An Unidentified, Filtrable Agent Isolated from the Feces of Children with Paralysis, Science **108:** 61, 1948

12. Ford, F. R.: Diseases of the Nervous System in Infancy, Childhood and Adolescence, ed. 2, Springfield, Ill., Charles C Thomas, Publisher, 1945, pp. 512-515, 437-441.

methods. Serum neutralization tests in mice, using samples taken during the acute febrile illness and convalescence, were reported as negative [2] for this disease by the Virus Laboratory of the United States Public Health Service.

Results of serum neutralization tests performed by the Bethesda virus laboratory for the identification of the St. Louis encephalitis virus were also negative.[2] This virus generally causes severe and diffuse involvement of the central nervous system,[12] but the possibility that the infection in our 18 cases was the abortive type of St. Louis encephalitis could not have been ruled out except by laboratory methods.

Meningoencephalitis, a frequent complication of the mumps virus,[13] may occur without parotid swelling and is commonly the exact diagnosis of random and undiagnosed cases of aseptic meningitis. Mumps virus infection was ruled out by clinical course, absence of parotid involvement and negative exposure history of the patients. However, infection with this virus now can be diagnosed by direct virus isolation or by noting a rising antibody titer by complement fixation tests.[14] These tests were done by the Bethesda virus laboratory using serum from our patients, and results were reported as negative.[2]

Also briefly considered in the diagnosis, but easily ruled out because of negative exposure and the lack of cutaneous manifestations, were the exanthematous virus diseases, measles, chickenpox and herpes zoster.

CONCLUSION

The etiologic agent in these 18 cases of aseptic meningitis, by processes of elimination, is a new virus. All possible viruses included in the differential diagnosis were ruled out. It is our opinion that this virus is probably related to the poliomyelitis virus but possesses a different mosaic pattern of antigenicity. By means of cross neutralizaion tests, the new virus isolated from patients whose disease was diagnosed as nonparalytic poliomyelitis or aseptic meningitis by Melnick, Shaw and Curnen [10a] is shown to be directly related to the virus in our series of cases. Although the Yale group of investigators state that a new suckling mice myositis virus was obtained from human sources because of the positive protection tests with human serums and the isolation of the virus more than once from the same material, there is no absolute proof that it is the causative agent of the disease. This could be determined only by inoculation of human volunteers with the virus.

SUMMARY

A series of 18 patients with aseptic meningitis admitted to the Charles V. Chapin Hospital in the autumn of 1948 is reported.

These patients experienced a short febrile illness characterized by meningeal irritation, severe headaches, minimal cervical stiffness, a lymphocytic pleocytosis in the spinal fluid and a favorable, uncomplicated, complete recovery with only symptomatic therapy.

The etiologic agent, a new virus, is determined by process of exclusion on the basis of clinical evaluation and virus studies performed by the virus laboratories at Yale University Medical School and at Bethesda, Md.

A similar, related virus entity has been reecntly observed by others.[15]

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EFFECT OF OBSTETRIC DIFFICULTIES AND MATERNAL DISEASE ON PREMATURE INFANT MORTALITY

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Several studies made by others [1] have stated the conclusion that maternal factors were not important in relation to the neonatal survival of the premature infant. The usual contention is that the birth weight of the premature infant largely determines its ability to initiate a satisfactory extrauterine existence. Obviously, birth weight as an evidence of maturity has a great deal to do with fetal survival. However, it is not the only controlling element in a satisfactory fetal prognosis. We have found the good health of the mother and the absence of serious obstetric complications to be of extreme importance. Only one other study, that of Diddle and Plass,[2] has indicated that factors other than birth weight influence fetal prognosis.

This paper is a review of our experience with premature infants born on our obstetric services from July 1, 1946 to Feb. 1, 1949. Among 477 premature births, in which the infants weighed between 1,000 and 2,500 Gm., the total fetal mortality was 16 per cent (table 1). This percentage includes stillbirths and neonatal deaths and is uncorrected for congenital anomalies. Premature infants from uncomplicated pregnancies and labors had but an 8 per cent mortality compared to a 37 per cent mortality when the pregnancy or labor was complicated (table 1).

The data represent the results obtained from our two teaching services, the Denver General Hospital and the Colorado General Hospital. During the thirty-one months from July 1946 to February 1949 there were 3,625 deliveries, of which 2,054 were at the Colorado General Hospital and 1,571 were at the Denver General Hospital.

INCIDENCE OF PREMATURITY

The incidence of births of infants in the 1,000 to 2,500 Gm. bracket was 13.2 per cent of our total. The proportion of live infants born weighing under 2,500 Gm. to total live births was 13.3 per cent. In reporting, 13.3 per cent would be known as our incidence of prematurity. Koch, Weymuller and James,[3] reporting for Long Island College Hospital, gave a 7 per cent incidence of prematurity. From 1930 through 1944 the incidence of prematurity at the Lying-In Hospital in Philadelphia was 9.4 per cent.[4] Eastman, of Johns Hopkins,[5] reported 10.7 per cent premature births. The number of infants born weighing less than 2,500 Gm. depends on several factors, such as the incidence of complications in pregnancy, the number of

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Read before the Section on Obstetrics and Gynecology at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 9, 1949.

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4. Tyson, R. M.: A Fifteen-Year Study of Prematurity, J. Pediat. **28**: 648, 1946.

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indigent mothers and the race. Although Negro patients make up 15 per cent of our clinic volume of cases, the incidence of prematurity among Negroes (9 per cent) was less than the general average for clinic patients. Thirty-four per cent of our deliveries were among Spanish-American indigent patients. Their incidence of prematurity, if judged by infants weighing less than 2,500 Gm., was 15 per cent. We are certain, however, that many Spanish-American indigent patients produce mature infants who weigh less than 2,500 Gm. at birth. This is not true of Spanish-American mothers in higher income groups.[6]

of mothers having no complications other than premature expulsion of a fetus weighing less than 2,500 Gm. The infant was born from a vertex position in each instance. Premature rupture of the membranes was not considered a complication if intrapartum infection did not occur.

The other large group of premature infants was born of mothers with severe uncontrolled medical or obstetric complications.

In this study controlled heart disease, arrested pyelitis and controlled diabetes were not considered as important related factors in fetal survival, and patients who

TABLE 1.—*Premature Mortality, 1,000 to 2,500 Gm., Divided into Complicated and Uncomplicated Pregnancies*

	Total Cases Liveborn	Neonatal Deaths	Per Cent of Neonatal Mortality	Cases Stillborn	Infants Lost	Per Cent of Fetal Loss	Total Infants	Uncomplicated Cases of Fetal Death	Complicated Cases of Fetal Death
1,000 to 1,500 Gm.									
Uncomplicated	36	9	25	5	14	34	41	14	..
Complicated	21	13	62	6	19	70	27	..	19
1,500 to 2,000 Gm.									
Uncomplicated	60	4	7	4	8	13	64	8	..
Complicated	41	11	27	6	17	36	47	..	17
2,000 to 2,500 Gm.									
Uncomplicated	237	3	1	2	5	2	239	5	..
Complicated	51	5	10	8	13	22	59	..	13
Total	446	45	10	31	76	16	477	27	49

Uncomplicated cases, 344; fetal deaths, 27; mortality, 8 per cent. Complicated cases, 133; fetal deaths, 49; mortality, 37 per cent.

TABLE 2.—*Distribution of Complicated Cases and Premature Neonatal Results (1,000 to 2,500 Gm.) for Complicated and Uncomplicated Pregnancies*

	Infants, 1,000 to 1,500 Gm.		Infants, 1,500 to 2,000 Gm.		Infants, 2,000 to 2,500 Gm.		Live Births, Totals	
	Lived	Neonatal Deaths	Lived	Neonatal Deaths	Lived	Neonatal Deaths	Lived	Died
Maternal complications *								
Eclampsia	1	..	..	1	..	1	1	2
Nonconvulsive toxemia	..	5	11	1	14	..	25	6
Placenta previa	2	1	6	..	5	..	13	1
Placental separation incomplete	2	2	4	2	2	..	8	4
Acute pyelitis	..	1	1	..	6	..	7	1
Breech presentation	3	2	8	1	14	2	25	5
Tuberculosis, active	..	..	..	..	3	..	3	..
Heart failure	..	..	..	..	2	..	2	..
Precip. delivery	..	1	..	..	..	1	..	2
Intrapartum amnionitis	..	..	..	2	..	..	..	2
Subacute bact. endocarditis	..	..	..	1	..	..	..	1
Syphilis, untreated	..	1	..	..	..	..	..	1
Uncontrolled diabetes	..	..	..	..	..	1	..	1
Cancer of the cervix	..	..	..	1	..	..	..	1
Face presentation	..	..	..	2	..	..	..	2
Total	8	13	30	11	46	5	84	29
Uncomplicated pregnancies †	27	9	56	4	234	3	317	16

* Complicated live births, 113; fetal deaths, 29; neonatal mortality, 26 per cent.
† Uncomplicated live births, 333; fetal deaths, 16; neonatal mortality, 5 per cent.

Our clinic incidence of toxemia was only 6 per cent, so this maternal condition probably does not make our incidence of small babies higher. Since over 98 per cent of the obstetric patients in both hospitals are on the ward service, we think that indigency, with all it implies in incomplete antepartum care, poor housing and subnormal diet, accounts for the high incidence of birth weights below 2,500 Gm.

METHOD OF STUDY

All cases have been separated into two large groups (tables 2 and 3). One group consists of those infants weighing between 1,000 and 2,500 Gm. who were born had such diseases were grouped with the normal mothers unless there was some obstetric complication.

NEONATAL MORTALITY

Our neonatal mortality rate for the first month of life among 480 consecutive live infants weighing less than 2,500 Gm. was 16 per cent. This compares favorably with reports from other teaching institutions.[7] The distribution of our patients into weight groups has been the same as that reported by others. In the 288 cases in the group from 2,000 to 2,500 Gm. the neonatal mortality rate was only 3 per cent. From 1,500 to 2,000 Gm. the neonatal mortality rate was

6. Goldstein, M. S.: Infants of Mexican Descent: I. Physical Status of Neonates, Child Development **18**: 3, 1947.

7. Dunham, E. C.: Premature Infants, Federal Security Agency, Children's Bureau Publications, no. 325, 1948, p. 42.

15 per cent. In the group from 1,000 to 1,500 Gm. 38 per cent of the infants died in the nursery. Ninety-one per cent under 1,000 Gm. failed to survive (table 4). Birth weight as an evidence of maturity has a great deal to do with fetal survival. However, it is not the only controlling element in survival. We have noticed that maternal complications influence fetal prognosis to a definite degree. Other authors have not reported the importance of maternal factors in relation to premature fetal survival. The exception to this is the work of Diddle and Plass, who showed that their

TABLE 3.—*Tabulation of Premature Stillbirths With and Without Maternal Complications*

	Infants, 1,000 to 1,500 Gm.	Infants, 1,500 to 2,000 Gm.	Infants, 2,000 to 2,500 Gm.
Associated with maternal complications *			
Bulbar poliomyelitis	..	..	1
Placental separation	..	2	2
Severe nutritional anemia	..	1	..
Eclampsia	1	2	..
Nonconvulsive toxemia	1	..	1
Erythroblastosis	..	..	2
Breech presentation	1	..	..
Thyrotoxicosis	..	..	1
Prolapsed cord	1	1	..
Placenta previa	1	..	..
Pneumonia	1	..	..
Auto accident and shock	..	..	1
Total	6	6	8
Not associated with maternal complications †			
Anencephalia	3	1	1
Normal pregnancy and labor	2	3	1
Total	5	4	2

Total stillbirths, 31.
* Stillbirths associated with maternal complications, 20 (65 per cent).
† Stillbirths associated with no maternal complications, 11 (35 per cent).

TABLE 4.—*Distribution of Premature Live Births by Weight Groups and Neonatal Mortality Rate*

Birth Weight, Gm.	No. of Cases	Per Cent of Distribution	No. of Neonatal Deaths	Per Cent of Neonatal Mortality
2,000 to 2,500	288	60	8	3
1,500 to 2,000	101	21	15	15
1,000 to 1,500	57	12	22	38
Under 1,000	34	7	31	91
Total	480	100	76	16

premature fetal mortality was two and one-half times greater in complicated cases than in noncomplicated cases.[2]

RESULTS OF THE STUDY

We have found that, given a healthy mother with none of the serious obstetric complications, our total fetal loss among infants weighing between 1,000 and 2,500 Gm. was only 8 per cent (table 1). Should the mother suffer from severe maternal disease, such as toxemia, placenta previa, placental separation or malpresentation (breech or face), our fetal mortality was 37 per cent, almost five times as great as in the noncomplicated cases.

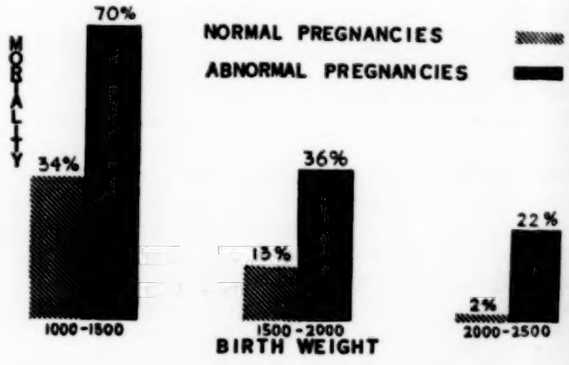
When we studied the neonatal death rate among premature liveborn infants we found parallel results that were consistent for each weight group. The neonatal death rate among infants from complicated pregnancies was 26 per cent. When the premature infant was born by vertex position from an uncomplicated pregnancy, the neonatal death rate was only 5 per cent.

When fetal loss among premature infants was studied within the various weight groups the following data were obtained (table 1 and the accompanying figure).

1. Infants of 1,000 to 1,500 Gm. from normal mothers had a 34 per cent fetal mortality (stillbirth and neonatal). Mothers with severe pathologic conditions lost 70 per cent of their infants in this group. The neonatal fetal mortality was 25 per cent in the normal cases and 62 per cent in the pathologic cases.

2. In the group weighing 1,500 to 2,000 Gm., normal mothers lost only 13 per cent of their infants. If the pregnancy was complicated 36 per cent of infants failed to survive. Neonatal mortality rates for the group weighing 1,500 to 2,000 Gm. were 7 per cent for normal cases, 27 per cent for abnormal cases.

3. In the group weighing 2,000 to 2,500 Gm. the total fetal loss was only 2 per cent in normal cases and 22 per cent in cases in which the pregnancy of the mother was complicated. Neonatal death occurred in less than 1 per cent of cases in



Comparison of premature fetal mortality rate in normal and abnormal pregnancies.

which the infants born of normal mothers weighed between 2,000 to 2,500 Gm. If maternal complications occurred there was a 10 per cent neonatal loss in this group.

The significant differences in premature deaths held true for our Denver General Hospital service as well as the Colorado General Hospital service. Table 5 is a presentation of the statistical significance of differences in the outcome of births following both complicated and uncomplicated pregnancies. These calculations show that our data are statistically significant.

MANAGEMENT OF UNCOMPLICATED PREMATURE LABOR AND DELIVERY FOR THE INTERESTS OF THE CHILD

Since the premature infant's ability to survive depends largely on its weight at birth and the absence of severe maternal disease or serious obstetric complications, the problem of prematurity for the present resolves itself into the prevention or control of factors that lead to this condition, the application of the best obstetric methods for delivery of the premature infant and the application of the best known methods for the care of the premature newborn child. Our method of approach has been the establishment of a Premature Infant Center at the University of Colorado as a combined training and service project of the Colorado State

Health Department and the university, with funds supplied by the United States Children's Bureau.[8]

When we have called our pediatricians to care for newborn premature infants (1,000 to 2,500 Gm.) from healthy mothers, only 1 in 20 have failed to survive. If the pregnancies were complicated seriously, 1 in 4 premature infants died during the first month of life (table 2).

The principles for management of a premature labor, uncomplicated by factors other than premature expulsion of the fetus in the vertex position, have been enumerated elsewhere.[9] Our routine is to give no form of analgesia or anesthesia other than local or low spinal anesthesia. Vitamin K is administered antepartum to the mother and immediately after birth to the child. Forceps rotations or midforceps operations are not done in these cases. Spontaneous undelayed termination of the second stage of labor is desired. Routine episiotomy for prevention of trauma to the fetal head is performed. If there is delay in the second stage of labor a gentle low or outlet forceps delivery is performed. As soon as the infant is born it is placed in a previously warmed incubator. A member of the pediatrics staff is in attendance for all premature deliveries. Gentle resuscitation is carried out by a person qualified in this art.

All babies who weigh under 2,000 Gm. at birth or who tend to become cyanotic are put in an incubator containing approximately 50 per cent oxygen. Those infants weighing under 1,500 Gm. are given additional 100 per cent oxygen through a funnel placed near the child's mouth. No attempt is made to feed an infant who has respiratory difficulties during the first forty-eight to seventy-two hours of life. Expert nurses, experienced in the care of premature infants, are in constant attendance. All infants suspected of infection or with respiratory difficulties receive prophylactic administration of penicillin from birth until they are no longer liable to sepsis. The uncomplicated pregnancies and labors discussed here have provided two thirds of the cases but only one third of the premature fatalities.

MANAGEMENT OF COMPLICATIONS OF PREGNANCY IN RELATION TO FETAL SURVIVAL

From our data we believe that in order to improve premature fetal survival more effort must be concentrated on the complicated cases. One third of the mothers in our series who had complications provided two thirds of the fetal deaths. For cases of toxemia of pregnancy, placenta previa, premature rupture of the membranes and breech delivery there are policies and procedures that may materially add to premature fetal survival if practiced. These policies have two aims: prolongation of gestation and protection of the fetus against such dangers as anoxia, obstetric trauma and infection. To accomplish these, the best hospital facilities, blood banks and careful professional judgment must be used. Prolonged antepartum hospitalization is often mandatory for the health of the mother and the unborn child. Transfusable blood must be readily available. The experience of an obstetric consultant should be used during the antepartum period, and an experienced pediatrician should be on hand at delivery so that specialized treatment of the premature infant can start at birth. Premature infants born from mothers with obstetric complications need all the specialized attention possible. If the hospital where delivery occurred is unable to provide such facilities, the infants should be transported to points where the highest type of care is available.

TABLE 5.—*Statistical Significance of Differences in Outcome of Births Following Complicated and Uncomplicated Pregnancies*

Birth Weight, Gm.	Comparison of Outcome as Related to Type of Pregnancy	Rate, Per Cent *	Difference Between Rates	Statistical Significance of Differences †		
				Not Significant	Significant	Highly Significant
1,000 to 2,500	Fetal loss					
	Complicated	37	29			x
	Uncomplicated	8				
	Neonatal mortality					
	Complicated	26	21			x
	Uncomplicated	5				
1,000 to 1,500	Fetal loss					
	Complicated	70	36			x
	Uncomplicated	34				
	Neonatal mortality					
	Complicated	62	37			x
	Uncomplicated	25				
1,500 to 2,000	Fetal loss					
	Complicated	36	23			x
	Uncomplicated	13				
	Neonatal mortality					
	Complicated	27	20			x
	Uncomplicated	7				
2,000 to 2,500	Fetal loss					
	Complicated	22	20			x
	Uncomplicated	2				
	Neonatal mortality					
	Complicated	10	9		x	
	Uncomplicated	1				

Calculations made by Dr. H. J. Dodge, Department of Public Health and Preventive Medicine, University of Colorado Medical Center.

* Calculation of rates:

(*a*) Per cent of fetal loss = $\frac{\text{Number of stillbirths + number of neonatal deaths} \times 100}{\text{Total births}}$

(*b*) Per cent of neonatal mortality = $\frac{\text{number of neonatal deaths} \times 100}{\text{Total live births}}$

† Statistical significance:

(*a*) Test of significance = difference in percentage ÷ standard error of difference in percentage.

(*b*) Formula for standard error = $\pm\sqrt{\frac{p_1 \times q_1}{N_1} + \frac{p_2 \times q_2}{N_2}}$

where subscripts 1 and 2 refer to the samples being compared, p equals the percentage occurrence of one outcome (death), q equals the percentage occurrence of the other outcome (survival), and N equals the total number of items in the sample.

(*c*) Level of significance:

Not significant—chance could account for a difference as great or greater more frequently than 0.05 of times.

Significant—chance could account for a difference as great or greater between 0.05 and 0.01 times.

Highly significant—chance could account for a difference as great or greater less frequently than 0.01 of repetitions.

Toxemias of Pregnancy.—The first step in reducing deaths from prematurity should be the elimination of toxemia of pregnancy. Thirteen (17 per cent) of our 76 premature fetal deaths were associated with toxemia of pregnancy. Twenty-three per cent of the premature babies born from toxemic mothers died. In our present state of knowledge, toxemia of pregnancy cannot be prevented but it can be controlled. By controlling the severity of toxemia, fetal survival can be increased. The

8. Taylor, E. S., and Gordon, H .H.: The Premature Infant Program in Colorado, The Mother **10**: 5, 1948.

9. (*a*) Beck, A. C.: How Can the Obstetrician Aid in Reducing Mortality of Prematurely Born Infants? Am. J. Obst. & Gynec. **42**: 355, 1941. (*b*) Beck, A. C.: The Obstetrician's Responsibility for the Hazards of the First Few Days of Life, with Special Reference to Anoxia and Prematurity, ibid. **51**: 173, 1946. (*c*) Taylor, E. S.: Obstetrical Aspects Concerning the Prevention of Neonatal Deaths, Rocky Mount. M. J. **45**: 211, 1948. Eastman.[5]

welfare of both mother and fetus must be considered in the management of these cases. Cosgrove [10] has expressed the opinion that until the thirty-fourth week of gestation physicians are justified in delaying the interruption of pregnancy in a toxemic patient for the benefit of the fetus, if the life of the mother is not in jeopardy. After the thirty-fourth week he considered that there is nothing to be gained by way of improving fetal prognosis, since after that time placental separation or intrauterine death will nullify any gain in maturity. In fact, neonatal fetal prognosis becomes worse after the thirty-fourth week. After two to three weeks of toxemic albuminuria the life of the mother may be endangered by continuation of the pregnancy.

Placenta Previa.—An expectant policy for treatment in certain cases of placenta previa has been recommended by others and has been adopted by us.[11] This policy applies to cases in which there is painless, causeless bleeding that occurs before the thirty-fourth week of gestation. Instead of proceeding with definitive treatment immediately in early cases (before the thirty-fourth week) of suspected placenta previa, we hospitalize these patients and prescribe rest in bed until six weeks from term. During the interim, suitably typed and cross-matched blood is made available. The cervix is inspected but not palpated, and rectal or vaginal examinations are not made. If labor ensues or if severe bleeding recurs, the expectant policy must come to a halt. Such a method of treatment will improve fetal survival.

Premature Rupture of Membranes.—Many premature deliveries are heralded by premature rupture of the membranes. When this takes place, we hospitalize the patient. If the rupture has occurred before the thirty-sixth gestational week we make no effort to induce labor. The patient is given 1 Gm. of sulfadiazine every four hours and 300,000 units of intramuscularly administered penicillin in oil daily. This is to prevent an intrapartum infection of the infant, membranes and fluid . If after three days the patient is not in labor and if the cervix appears unripe, we may send her home. The oral administraion of sulfadiazine and penicillin is continued. Instructions in perineal hygiene are given. By this method, gestation has been prolonged for the benefit of the child for periods varying from a week to a month. When the infant is born, prophylactic penicillin and sulfadiazine therapy should be given the child in the nursery.

Breech Delivery.—Premature fetal survival is jeopardized by breech delivery. The relatively large size of the fetal head over shoulder diameter as discussed by Beck [9b] leads to cervical dystocia and intracranial hemorrhage. Our neonatal premature mortality (infants weighing 1,000 to 2,500 Gm.) in breech cases has been 20 per cent. Skill must be exerted in the breech delivery of a premature infant. If there is any obstruction the physician should be prepared to do Dührssens' incisions of the cervix to prevent trauma to the after-coming fetal head as it attempts to negotiate the incompletely dilated cervix. The application of suprapubic pressure to the after-coming head by a vigorous assistant is condemned. Analgesia and general anesthesia should be withheld.

10. Cosgrove, S. A.: The Clinical Management of Pre-Eclampsia and Eclampsia, Bull. Margaret Hague Maternity Hosp. **1**: 40, 1948.

11. Johnson, H. W.: The Conservative Management of Some Varieties of Placenta Previa, Am. J. Obst. & Gynec. **50**: 248, 1945. Macafee, C. G. H.: Placenta Previa-Study of One Hundred and Seventy-Four Cases, J. Obst. & Gynaec. Brit. Emp. **52**: 313, 1945. Jeffcoate, T. N. A.: Placenta Previa, Practitioner **159**: 15, 1947. Williams, T. J.: The Expectant Management of Placenta Previa, Am. J. Obst. & Gynec. **55**: 169, 1948.

SUMMARY

This is a study of 76 infant mortalities among 477 premature births. In addition to the presentation of results obtained in the preservation of the premature infant's life, mortality has been correlated with serious maternal diseases and serious obstetric difficulties. Previous studies by others have concluded that the survival of the premature infant is largely dependent on its weight at birth. This study agrees in part with this conclusion but shows that maternal conditions such as toxemia, placenta previa, other placental pathologic conditions, breech presentation and maternal infections influence the prognosis of the premature infant to a definite degree.

In premature infants born of mothers without complicating factors other than premature delivery there was an 8 per cent mortality rate. This rate was about one fifth that of premature infants born of mothers who had serious medical and obstetric complications.

When the premature infant was born by vertex position from an uncomplicated pregnancy, the neonatal death rate was only 5 per cent. The neonatal death rate among infants from complicated pregnancies was 26 per cent. The difference in the neonatal mortality rate demonstrated between normal and abnormal pregnancies was consistent for all weight groups.

Since maternal complications contribute so heavily to premature infant mortality, obstetric management of complications for the best interests of both mother and child is emphasized.

CONCLUSIONS

1. In our series premature infants born of mothers suffering from severe uncontrolled medical disease or of mothers having serious obstetric complications have a mortality rate four to five times that of infants born of healthy mothers without complications of pregnancy.

2. Previous literature and previous concepts have held that maternal complications do not materially influence premature fetal survival.

3. Birth weight, as others have emphasized, is important in premature fetal survival, but such conditions as maternal infections, toxemia of pregnancy, placenta previa, placental separation and malpresentations also affect the neonatal mortality of the premature infant.

4. The large responsibility of improving the mortality rates of premature infants rests with those doing obstetric work since two thirds of premature fetal deaths are associated with maternal complications.

5. Many maternal complications, if they cannot be prevented, can be controlled to improve fetal prognosis.

6. Through special attention to the premature infant by our obstetricians, pediatricians and nurses, the neonatal premature fetal mortality in two hospitals for the indigent (Denver General Hospital and Colorado General Hospital) has been 16 per cent of all live infants under 2,500 Gm.

CONDUCTION ANESTHESIA

Protection Afforded the Premature Infant

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The lowest uncorrected premature infant mortality rate reported has been attained at the St. Louis Maternity Hospital during a recent three and one-half year period because of the protective effect of conduction (continuous caudal or saddle block) analgesia and anesthesia. This study has been made with the cooperation of the Departments of Obstetrics and Pediatrics of the Washington University School of Medicine, and the results of these joint efforts are of major importance in the field of obstetric anesthesia.

From July 10, 1945 until Dec. 27, 1948, 3,000 labors and deliveries were completed with the use of conduction anesthesia. Of the 3,000 patients in labor, 2,500 received caudal and 500 saddle block anesthesia. The conduction anesthesia series consisted of 28.2 per cent of the 10,640 deliveries that occurred during the control period. This series included 631 premature infants, an incidence of 5.9 per cent. This figure agrees generally with those previously noted by Adair [1] and by Dippel and associates [2] but is somewhat below that reported by Tyson.[3] There were 40 macerated stillborn infants in the series. With the exception of their use in establishing the incidence of premature births, they were immediately eliminated from statistical consideration, leaving a total of 591 premature births available for analysis.

It is not the province of this paper to discuss the technics or the indications and contraindications of caudal or saddle block analgesia. These considerations have appeared in voluminous detail in the literature since the first giant strides in the field of conduction anesthesia for obstetrics were made by such men as Hingson,[4] Lundy [5] and Adriani.[6] It is sufficient to say that we have had no maternal deaths and minimal maternal morbidity attributable to conduction anesthesia in the 3,000 cases under consideration.

What constitutes a premature labor and delivery? Is the prematurity of an infant to be determined in terms of its weight at birth, its length at birth or its estimated duration of intrauterine existence? Hess and Chamberlain [7] have claimed that all three factors are of major importance and must be considered in unison. Dippel and co-workers [2] have suggested the combination of an additional number of variants in order to arrive at a premature survival index. The policy at the St. Louis Maternity Hospital has been to indicate prematurity purely on a weight basis, although admiting the obvious inadequacies of this delineating system (the smaller twin in a term pregnancy, for example). We have chosen this standard because it offers the best method of comparison with previously established results reported from other clinics.

METHODS OF STUDY

The care of all the premature infants in this series, once these infants reached the nursery for premature babies, was under the complete supervision and control of the Department of Pediatrics of Washington University. All premature infants admitted to the nursery were cared for by identical staff personnel, senior staff physicians, house officers and nurses without any indication as to the method of analgesia or anesthesia employed for the mother. The infants were treated in an identical manner (within a normal variant) as to medication and methods of therapy in a conscientious effort to provide, so far as possible, a well regulated clinical control series.

It was impossible to assign alternate cases of premature labor to conduction anesthesia when these technics were first introduced at the St. Louis Maternity Hospital, because these methods were then still in the stage of experimental development. As a result, about 70 per cent of the premature labors reported as comprising the nonconduction group are from the private service of the hospital, and the corresponding 70 per cent in the conduction group are from the ward service.

The nonconduction group consists of those patients whose labors and deliveries were carried out under twilight sleep (morphine-scopolamine anesthesia), inhalation anesthesia, pudendal block, thiopental sodium (pentothal sodium®) anesthesia or any combination of these methods. Also included in this group are the 17 patients whose labors and deliveries were carried out without any analgesia or anesthesia. This group had an excessively high mortality rate, with 7 infant deaths out of the 17 deliveries. The reason for including patients with pudendal block and those without any analgesia or anesthesia in the nonconduction group will be presented later in the paper.

A total of 347 premature infants comprise the nonconduction group and 244 the conduction group. The first 1,500 labors and deliveries in which caudal anesthesia was used have been previously reported.[8] This paper will review in more detail some features of the earlier observations and add the results obtained in an additional 1,000 deliveries with caudal anesthesia and 500 deliveries with saddle block anesthesia.

The control period of the first 1,500 deliveries with conduction anesthesias encompassed about two years (table 1). It was during this period that the technics of caudal anesthesia were developed, and the work progressed slowly at first. The second period of 1,500 deliveries with conduction anesthesia (table 2) required approximately one-half year less to complete because of the increased familiarity on the part of the house physicians with the technics of conduction anesthesia and its resultant increased application to a larger number of patients. The decision to divide the premature infants into 500 Gm. weight groups was made primarily to demonstrate more clearly the tremendous increase in premature deaths in infants below 1,500 Gm. Pediatricians throughout the country have emphasized the fact that the infant weighing less than 1,500 Gm. presents an entirely different problem in basic metabolism than the premature infant at higher weight levels.

Read before the Section on Obstetrics and Gynecology at the Ninety-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 9, 1949.

1. Adair, F. L.: Obstetrics and Gynecology, Philadelphia, Lea & Febiger, 1940, vol. 1.
2. Dippel, A. L.; Johnson, H. W., and Cornelison, J. L.: Premature Survival Index, Am. J. Obst. & Gynec. **54**: 1004, 1947.
3. Tyson, R. M.: A Fifteen Year Study of Prematurity, J. Pediat. **28**: 648, 1946.
4. Edwards, W. B., and Hingson, R. A.: Continuous Caudal Anesthesia in Obstetrics, Am. J. Surg. **57**: 459, 1942.
5. Adams, R. C.; Lundy, J. S., and Sheldon, T. H.: Continuous Caudal Anesthesia or Analgesia, J. A. M. A. **122**: 152 (May 15) 1943.
6. Adriani, J., and Roman-Vega: Saddle Block Anaesthesia, Am. J. Surg. **71**: 12, 1946.
7. Hess, J. H., and Chamberlain, I. M.: Premature Infants: A Report of Two Hundred and Sixty-Six Consecutive Cases, Am. J. Dis. Child. **34**: 571 (Oct.) 1927.
8. Masters, W. H.: Caudal Analgesia, Am. J. Obst. & Gynec. **56**: 756, 1948.

The statistics available in this series bear out that contention.

From an obstetric point of view the fetal deaths were cataloged into the headings of stillborn macerated, intranatal, postnatal and neonatal deaths. These arbitrary divisions were necessary to show more clearly

TABLE 1.—*Premature Infants Among 5,623 Deliveries Made with Patients Under Conduction and Nonconduction Anesthesia from July 10, 1945 to June 17, 1947 (4,123 Nonconduction, 1,500 Caudal)*

Weight Group, Gm.	Total No. in Group	Intranatal Deaths	Postnatal Deaths	Neonatal Deaths	Total Deaths	Per Cent of Infant Mortality
Nonconduction Anesthesia, 241 *						
2,500 to 2,001.....	152	4	2	6	12	7.8
2,000 to 1,501.....	48	2	0	8	10	20.8
1,500 to 1,001.....	25	4	1	6	11	44.0
1,000 to 500.....	16	8	4	4	16	100.0
Conduction Anesthesia, 109 †						
2,500 to 2,001.....	69	0	0	1	1	1.4
2,000 to 1,501.....	23	0	0	1	1	4.4
1,500 to 1,001.....	9	0	0	2	2	22.2
1,000 to 500.....	8	0	0	6	6	75.0

* Premature incidence in this group is 6.5 per cent (26 stillborn macerated infants included).
† Premature incidence in this group is 7.5 per cent (3 stillborn macerated infants included).

the real protection afforded by conduction anesthesia. Any infant with clinical evidence of viability (fetal movement or fetal heart tone) when labor began but who expired during the course of labor or delivery was termed an instance of intranatal death. If death occurred immediately after delivery, before the infant could be admitted to the nursery for premature infants and placed under the care of the pediatric staff, this fatality was arbitrarily listed as a postnatal death. Once the infant was admitted to the nursery for premature infants, mortality was considered under the

TABLE 2.—*Premature Infants Among 5,027 Deliveries Made with Conduction and Nonconduction Anesthesia from June 18, 1947 to December 27, 1948 (3,527 Nonconduction; 1,000 Caudal, and 500 Saddle Block)*

Weight Group, Gm.	Total No. in Group	Intranatal Deaths	Postnatal Deaths	Neonatal Deaths	Total Deaths	Percentage of Infant Mortality
Nonconduction Anesthesia, 106 *						
2,500 to 2,001.....	66	1	1	3	5	7.5
2,000 to 1,501.....	27	3	1	3	7	25.9
1,500 to 1,001.....	9	2	0	5	7	77.8
1,000 to 500.....	4	2	1	1	4	100.0
Conduction Anesthesia, 135 †						
2,500 to 2,001.....	69	1	0	3	4	5.8
2,000 to 1,501.....	42	0	1	2	3	7.1
1,500 to 1,001.....	19	0	0	5	5	26.3
1,000 to 500.....	5	1	0	3	4	80.0

* Premature incidence in this group is 3 per cent (9 stillborn macerated infants included).
† Premature incidence in this group is 9 per cent (2 stillborn macerated infants included).

heading of neonatal death. All premature infants were followed only until they were discharged from the nursery.

The statistics to be reviewed are of significance only if no attempt at selection or control of the cases, however unwitting, has been made by the operators. Previous publications [9] have detailed the true objectivity of the experiment by dividing the first control group in half (to aid in statistical analysis) and have shown that there is the expected constancy of results so necessary for a well controlled series. This work will not be reduplicated here. It was the constant concern of all connected with this control series to avoid any suggestion of selection of cases.

The one place where the objectivity of the control series failed was in its application to patients demonstrating medical obstetric complications. Since it is the feeling of physicians on the obstetric service that conduction anesthesia offers particular protection to those patients who enter labor with cardiorespiratory disease complicating the obstetric picture, virtually all the patients exhibiting organic heart disease or active pulmonary infection were delivered by conduction anesthesia methods. In addition, 85 per cent of patients with diabetes and 80 per cent of all patients with severe preeclampsia or eclampsia were also delivered under the protection of conduction anesthesia. Thus the

TABLE 3.—*Total Number of Premature Infants Among 10,650 Deliveries Made with Conduction and Nonconduction Anesthesia from July 10, 1945 to Dec. 27, 1948 (7,650 Nonconduction; 2,500 Caudal, 500 Saddle Block)*

Weight Group, Gm.	Total No. in Group	Intranatal Deaths	Postnatal Deaths	Neonatal Deaths	Total Deaths	Per Cent of Infant Mortality
Nonconduction Anesthesia, 347 *						
2,500 to 2,001.....	218	5	3	9	17	7.8
2,000 to 1,501.....	75	5	1	11	17	22.7
1,500 to 1,001.....	34	6	1	11	18	52.9
1,000 to 500.....	20	10	5	5	20	100.0
Total.....	347	26	10	36	72	20.8
Conduction Anesthesia, 244 †						
2,500 to 2,001.....	138	1	0	4	5	3.6
2,000 to 1,501.....	65	0	1	3	4	6.2
1,500 to 1,001.....	28	0	0	7	7	25.0
1,000 to 500.....	13	1	0	9	10	76.9
Total.....	244	2	1	23	26	10.7

* Premature incidence in this group is 4.9 per cent (35 stillborn macerated infants included).
† Premature incidence in this group is 8 per cent (5 stillborn macerated infants included).

only selection in the series was that made necessary by the feeling that those patients whose obstetric patterns were clouded by complicating disease patterns should be protected in labor by either caudal or saddle block anesthesia.

In the analysis of table 1, the major differences between the two groups are notably concentrated in the columns indicating intranatal and postnatal deaths. There were no fetal deaths among the 109 patients of the conduction group from the time the patient entered labor until the infant was admitted to the nursery for premature infants. The possible significance of this pronounced difference will be discussed in detail later. In the final column showing the percentage of infant mortality, the real difference in the total mortality is evident in the figures for lines which indicate comparable weight. The top weight group of 2,500 to 2,001 Gm. shows an excessive difference that is made more remarkable by the unusual figure of 1.4 per cent infant mortality in the conduction group. This figure is entirely too low, actually lower than the established expected fetal mortality of the term infant.[8] It is expected to rise with further continuation of the series.

9. Lull, C. B., and Hingson, R. A.: Control of Pain in Childbirth, Philadelphia, Lippincott, 1948. Masters.[8]

The same consideration may be expected to apply to a lesser extent in the 2,000 to 1,501 Gm. group. Of major importance is the continued lowered mortality rate in the 1,500 to 1,001 Gm. group. The essential equality of premature incidence in the two control groups is notable. In the subsequent series (table 2) the continued excellent results in the conduction group stimulated staff physicians to an increased administration of conduction anesthesia to women in premature labor. This resulted in an excessive premature incidence in the second control period of the conduction series.

The second control period was also carried out for the length of time necessary to complete 1,500 labors and deliveries under conduction anesthesia. During this series saddle block anesthesia was introduced to the St. Louis Maternity Hospital, and the technic of the house physicians who conducted all 3,000 conduction anesthesia labors was improved and perfected. For this reason the second conduction group consists of 500 deliveries using saddle block anesthesia in addition to 1,000 deliveries using caudal anesthesia. Table 2 indicates a decided change in the total number of infants in the groups under consideration. There are now as many premature infants in the conduction group as in the nonconduction group. This change, as suggested previously, is the result of the maintained excellence of results in the conduction group and the resultant increase in the popularity of this method of anesthesia in premature labor and delivery. There was also a general increase in the popularity of conduction methods. Because of this popularity, the total of 1,500 cases was achieved in approximately six months less time than that taken by the first series.

The basic comparison in the columns indicating intranatal and postnatal deaths continues in the same vein previously noted in table 1. In the final column (percentage of infant mortality), the differences continue as described. In the 2,500 to 2,001 Gm. group the conduction mortality increased, as had been expected. The low mortality rates at the 2,000 to 1,501 and 1,500 to 1,001 Gm. levels for the conduction groups continue at the same low percentage figures found in the first half of the series. The premature incidence of 3 per cent in the nonconduction group and an excessively high 9 per cent incidence in the conduction group is due to the realization that conduction analgesia increased the chance of the premature infant for survival.

Table 3 is a complete summation of the three and one-half years' work. It provides a basis for easy comparison of data, which will be discussed in detail.

COMMENT

The demand for conduction anesthesia as a major weapon in the armamentarium of the practicing obstetrician has grown rapidly during the past eight years. It has remained for clinical experience in the field to suggest its place of greatest value. The increased protection afforded the premature baby by conduction anesthesia is certainly of major importance. From a purely obstetric point of view, there is no differentiation between caudal and saddle block anesthesia as methods of conduction anesthesia. Both technics have identical approaches to the protection of the infant. They vary only from a neuroanatomic standpoint as far as the details of technic are concerned. The individual indications or contraindications are not the province of this paper. For these reasons and to insure clarity the two will be discussed as one.

Conduction anesthesia protects the premature infant both during and after delivery. Positive protection is provided by the alteration in the normal course of labor that inevitably accompanies any method which interferes with the control over labor asserted by the autonomic nervous system. The first stage of a labor in which the patient is under conduction anesthesia is somewhat slowed when compared to the similar stage under the influence of other analgesic agents or when unprotected with any medication. The premature head, with its soft skull cap and widely separated cranial sutures, responds unfavorably to the excessive pressures of the usually rapid labor associated with premature delivery. The respiratory center is depressed and tentorial tears and cerebral hemorrhages are much more frequent than in the term infant. As noted in table 3, 10 per cent of those infants without conduction anesthesia died either during the course of labor or immediately thereafter. It is possible that they died because they could not withstand the excessive pressures of generally rapid labor or delivery.

Many premature labors terminate in essentially precipitous deliveries despite the most diligent obstetric care. Thus the dangers of the greatly increased pressure coincident with rapid first and second stages of labor are augmented by the sudden release of pressure from the premature's head provided by a precipitous delivery. In all probability, this sudden release of pressure is even more serious in consequence than the sudden application of pressure. The precipitous release of pressure from the yielding premature head often results in cerebral edema, tentorial tears or hemorrhage. Occasionally the counterpart of the foregoing picture prevails: the premature head is pounded against the tight unyielding perineal body characteristically found in the nulliparous woman, and the infant, unable to withstand the rigors of such punishment, dies during the second stage of labor.

Under conduction anesthesia the labor progresses in a different manner. The tendencies toward precipitous labor or delivery disappear. The resistance of the perineal floor is essentially eliminated as the result of the extensive perineal relaxation concomitant with the use of conduction anesthesia. At the St. Louis Maternity Hospital as soon as any patient achieves complete dilatation of the cervix and the premature head reaches the perineal floor, the patient is immediately prepared for delivery. With the patient receiving 100 per cent oxygen as additional protection for the infant, the premature head is gently and easily guided over the relaxed perineal floor and delivered by forceps in an effort to reduce to the barest minimum the rigors of the second stage of labor. The indisputable proof that this method is effective in handling premature deliveries is evidenced by the fact that (table 3) only 3 of 244 premature infants who were alive when labor began died during the progress of the labor or delivery or before they could be admitted to the nursery for premature infants. In direct contrast, 36 of 347 premature infants died either during labor or immediately thereafter when other methods of analgesia were employed. Breech presentations and multiple births when the labor is premature are also delivered as soon as dilatation of the cervix is complete and the presenting part rests on the relaxed pelvic floor.

In addition to the positive protection afforded by conduction anesthesia to the premature infant during

labor, there are significant advantages which become apparent after delivery. Primarily, the labile premature respiratory center is provided with increased protection. The use of methods of conduction anesthesia obviates the need for premedication of the mother. Such respiratory depressants as the opiates or barbiturates have no place in premature labor, and the demand for the relief they provide vanishes when the patient is not distressed. In addition, the premature respiratory center is spared the effects of an inhalation anesthetic at delivery. There is no need to establish relative anoxia in the mother if the distress of delivery has been previously obviated by saddle block or caudal anesthesia.

Further evidence of a postdelivery type of protection afforded the premature infant was evidenced by the published figures [8] on the resuscitation rate of infants born under the protection of caudal analgesia. It was pointed out that the resuscitation rate for the entire premature component of the first control series (109 infants) was only 6 per cent. This figure was amazingly close to the record published by Irving and associates [10] of a 1.9 per cent resuscitation rate in normal term deliveries without anesthesia of any sort. The fact that the premature infant can be well oxygenated at delivery (100 per cent oxygen during the process) makes resuscitation an easier procedure and less of an emergency.

It should be pointed out that those patients (a total of 17) who were not given anesthesia or analgesia were also assigned to the nonconduction group. Although these patients were not given respiratory depressants for labor or delivery, this negative protection to the premature infant was more than counteracted by the loss of positive protection due to cerebral tension on the infant caused by the generally rapid labors. The greatly increased pressure of a second stage of labor with its concomitant reflex bearing down, on the part of the mother, which brings into play the powerful abdominal muscles, should be avoided whenever possible in the supervision of the premature labor and delivery.

We certainly consider pudendal block as a form of conduction anesthesia. However, those patients in the series whose premature labors and deliveries were carried out under this method (a total of 11) were also classified in the nonconduction group by a purely arbitrary ruling. The use of pudendal block is discouraged in premature labors primarily because it is believed that premature labor is one of the few contraindications to this technic. Although this method provides a relaxed perineum and avoids many of the dangers of the second stage of labor, it affords essentially no protection for the first stage of labor. In addition, premedication in varying amounts was used during the first stage of labor in all 11 cases under consideration, and the local block was applied only after the patients were well into the second stage of labor. If saddle block or caudal anesthesia were not available, pudendal block would be the treatment of choice in premature labor, but because of its obvious local application, pudendal block has not the advantages of complete protection for the premature labor that its companions in needle anesthesia provide.

Finally, credit must be given to the Department of Pediatrics and the staff of the nursery for premature infants. A search of the literature has revealed that the lowest figures for uncorrected premature mortality were published by Dippel and co-workers [2] at 22 per cent and by Tyson [3] at 26 per cent. A glance at table 3 will reveal the two most important results achieved in the entire program. They are the figure of 20.8 per cent as the uncorrected premature mortality of the nonconduction group and the unprecedented figure of 10.7 per cent as the uncorrected mortality in the conduction group. Stillborn infants were not included by Dippel or Tyson. However, since it is felt that any premature infant who is alive when labor begins should be accepted as a fatality by the obstetrician, only stillborn macerated infants were excluded from the mortality statistics. This makes these low mortality figures even more remarkable. It is obvious that as the result of the superior protection provided the premature infant during the neonatal period by the unexcelled work of the pediatric staff, the uncorrected premature mortality even in the nonconduction group reached a new low of 20.8 per cent. The additional protection afforded the premature infant by the use of conduction anesthesia has reduced the mortality of premature infants by at least 100 per cent over that of the nonconduction group. Thus, as the result of the combined efforts of members of the pediatric and obstetric staffs, an unprecedented figure for uncorrected premature mortality, 10.7 per cent, has been achieved over a three and one-half year control period.

SUMMARY

1. Members of the Pediatric and Obstetric staffs of the Washington University School of Medicine have cooperated to establish a control period in an effort to lower premature mortality.

2. Patients were divided into two groups, conduction and nonconduction, depending on the type of analgesia or anesthesia used during their labors and deliveries.

3. The nonconduction group consisted of patients whose labors and deliveries were carried out under twilight sleep (morphine-scopolamine anesthesia), inhalation anesthesia, pudendal block, thiopental sodium anesthesia or any combination of these methods. Those patients delivered without any analgesia or anesthesia were also included in this group.

4. The conduction group consisted of patients whose labors and deliveries were carried out under the influence of caudal or saddle block analgesia and anesthesia.

5. During the three and one-half year control period, divisions were made at 1,500 and 3,000 cases with conduction anesthesia for statistical analysis.

6. The infant mortality from labors protected by conduction anesthesia was consistently at least 100 per cent lower than that shown in the nonconduction group.

7. The protection provided the infant during labor and after delivery by conduction anesthesia and analgesia was described in detail.

8. As the result of the efforts of the combined staffs, an uncorrected infant mortality of 20.8 per cent was achieved for the nonconduction group. This figure is lower than that of any comparative series previously published.

9. In the conduction group the uncorrected infant mortality was 10.7 per cent. This figure represents a reduction in premature infant mortality of at least 100 per cent over any figure previously published.

630 South Kingshighway Boulevard.

10. Irving, F. C.; Berman, S., and Nelson, H. B.: The Barbiturates and Other Hypnotics in Labor, Surg., Gynec. & Obst. 58: 1, 1934.

ABSTRACT OF DISCUSSION

ON PAPERS BY DRS. TAYLOR, PHALEN AND DYER AND DRS. MASTERS AND ROSS

DR. FRANKLIN F. SNYDER, Cambridge, Mass.: Drs. Taylor, Phalen and Dyer made the observations that five times as many babies die in the presence of maternal complications as die in their absence; this illustrates the magnitude of maternal complications in premature labor. Two thirds of the fatalities in their series were associated with maternal complications. Drs. Masters and Ross showed the influence obstetric analgesia and anesthesia may have on mortality in premature labor. In this series the mortality was only half as great when caudal or low spinal anesthesia was used as when other methods were used. The mortality rate was reduced to the low level of 11 per cent. Explanation of these results is of great interest. However, it is not easy to dissociate the individual role of factors of great potency when all are operative at the same time. These factors include, as has been pointed out, pediatric care, obstetric judgment, elimination of narcosis, alteration of the mechanism of labor by medication, so that the summation of effects is favorable, and selection of cases. One approach to further clarification has been employed, and that is a comparison of the mortality rate after low spinal or caudal anesthesia with that after various other types. In the present series of cases in which nonconduction anesthesia was used, the conclusions to be drawn are limited by the fact that the results with individual methods such as morphine-scopolamine, thiopental sodium or inhalation anesthesia and pudendal block are given not separately but together. In tracing the action of anesthetic agents on the premature baby or in seeking the effects of maternal complications, there is the question as to the chief cause of death. Both clinical histories and autopsy observations support the conclusion that fatality is associated chiefly with functional injury rather than with structural damage. It is failure of the oxygen supply to the respiratory system, or asphyxia, that is most immediately concerned.

DR. ROBERT A. HINGSON, Baltimore: These reports confirm the reductions in premature infant mortality that have been reported by other large American hospitals during this decade. Better obstetric care, better pediatric care and better management of anesthesia have each shared in the credit for this success. There is no single factor responsible for such results. The Johns Hopkins Hospital electively uses conduction anesthesia in delivering 60 per cent of its premature babies. The hazards of maternal complications in accentuating premature mortality are recognized. The total uncorrected premature infant mortality (both stillbirths and neonatal deaths) of 16 per cent from the Denver report (Drs. Taylor, Phalen and Dyer) and of 21 per cent from the St. Louis report (Drs. Masters and Ross) rank among the best in the literature. The St. Louis report of 249 premature deliveries in which conduction anesthesia was used with an uncorrected infant mortality of 12.4 per cent represents the highest incidence of salvage of the premature infant on record. In both reports the careful analyses of groups of babies in the 500 Gm. weight categories add substantial and well organized information to the published records in obstetrics that will be of permanent assistance to other analysts in this field. It is regrettable that the Denver report did not include an analysis of infant mortality in the 500 to 1,000 Gm. group. The emphasis on the role of obstetric difficulties and maternal disease on premature infant mortality is well supported and documented. This factor suggests that a more adequate treatment and prevention of these maternal factors is the first line of attack in reducing the infant death rate. The incidence of 13.2 per cent premature deliveries in Denver and 6 per cent premature deliveries in St. Louis again emphasizes the importance of the premature infant in the nationwide problem of infant mortality. I concur with the opinions of the authors in recommending that systemic analgesia and general anesthesia be withheld from mothers who are delivering premature babies. It is my opinion at this time that the withholding of systemic analgesia and general anesthesia and the substitution of one of the properly executed forms of conduction anesthesia, reinforced by oxygen therapy, proper fluid and blood pressure balance, offers the premature baby its best chance for survival.

DR. HARRY M. KIRSCHBAUM, Detroit: I am sorry to disagree with these beautiful presentations, but there are some truths that must be brought out. Dr. Snyder did a monumental piece of work. He did not, however, have a chance to emphasize the point that any drug except scopolamine will depress the respiration. Dr. Masters called attention to the fact that patients were given pentobarbital sodium or thiopental sodium during labor and then were delivered with the use of a general anesthetic. For that reason, he believed that the survival rate of the premature infant should compare with the conduction anesthesia rate. That would be impossible. If a physician removes the gas mask or ether mask from the face of the patient and lets her breathe some air just as he is bringing the head of the child over the perineum, he will find the results about equal, and he will not have the necessity of endangering the mother with caudal anesthesia. At Cedars of Lebanon Hospital in Los Angeles there are six teams of conduction anesthetists. That would be difficult in the ordinary hospital. I think that the death rate due to the anesthesia at this hospital was something like 3 in 3,000 cases. The total death loss was 7, but only 3 deaths were attributed to caudal anesthesia. I prefer to administer scopolamine throughout the labor, beginning as soon as labor commences. Then at the end of labor I prefer to inject 30 or 40 mg. of procaine hydrochloride intraspinally to carry out the actual delivery. Since premature infants die from atelectasis or cerebral hemorrhage, I give all patients 5 mg. of vitamin K daily during the last three months of pregnancy and an intravenous injection of vitamin K on entrance to the hospital. This dose is repeated in eight hours if labor is prolonged. Scopolamine alone plus a spinal injection of procaine hydrochloride, 30 to 40 mg., is administered for the labor and delivery.

DR. CHARLES A. WEYMULLER, Brooklyn: I have the privilege of reporting a series of almost the same size as that of Dr. Taylor from the Long Island College Hospital, compiled by Dr. Carl Tafeen of the obstetric service. Our series also represents a five year period and involves the group of premature infants described before the American Medical Association in Atlantic City, N. J., by Koch, James and me in July 1947. We reported 453 premature infants as compared with 477 in the Denver report (Drs. Taylor, Phalen and Dyer); their mortality was 16 per cent, and ours was 18 per cent. In uncomplicated pregnancies, we reported 298 premature births against their 344. The mortality rates were almost identical (9 per cent against 8 per cent). In complicated pregnancies, Long Island College Hospital had 155 against 133 from the Denver hospitals. Both groups had a mortality rate of 37 per cent. Therefore, if Dr. Taylor elects to add our group to his, the total would rise from 477 cases to 930, a really sizable group. Since our analyses of all infants, including those born prematurely, is complete for our previous 11,000 deliveries, we will soon add that series to this one. Thereby, the validity of this trend should receive a thorough testing. Further comparisons show the incidence of various complications of pregnancy and the mortality percentages for premature infants delivered under those individual circumstances. We had 134 cases of complicated pregnancy against 113 from the Denver hospitals; our mortality was 40 per cent, theirs 37 per cent. We found that three entities: mechanical obstetric complications, accidental hemorrhage and preeclamptic toxemias were operative in 87 of our 134 cases. In the Denver hospitals these factors were present in 81 of 113 cases. When obstetric complications were operative at Long Island College Hospital the mortality rate was 37 per cent and at the Denver hospitals 30 per cent; after accidental hemorrhage the Long Island College Hospital showed a 60 per cent mortality and the Denver hospitals 33 per cent; after preeclamptic toxemia the Long Island College Hospital had a mortality rate of 33 per cent and Denver 25 per cent. Our experiences were strikingly similar and our over-all mortality rates close.

DR. CARL H. TAFEEN, Brooklyn: It is clear from this study that a premature infant from an uncomplicated pregnancy has an extremely good chance of survival compared to an infant of similar or even greater weight from a complicated

pregnancy. Our emphasis must therefore be directed toward the latter if we hope to reduce fetal mortality in premature births. Dr. Taylor has directed his remarks toward a consideration of obstetric management among patients who have toxemia, placenta previa, breech delivery and premature rupture of membranes. Dr. Weymuller and I are in full accord. It has been established that a patient with placenta previa who is left alone does not die of initial hemorrhage. We are practicing the expectant treatment discussed by Dr. Taylor in all cases of placenta previa involving a small or premature infant. Even when delivery is planned, at least a few hours are allowed to elapse during which the lost blood is replaced, so that an infant is not born with the complication of anoxia added to that of prematurity. The figures in Dr. Taylor's series and in our series show that the breech delivery of a premature infant is synonymous with sharply increased fetal mortality. We still believe that all infants should be turned by external version as soon as they are viable. Many turn spontaneously in the latter weeks of gestation, but the procedure has proved to be so safe and simple that we do not await such an outcome. We certainly cannot draw any conclusions regarding the patient with premature rupture of the membranes, but we have been encouraged by following the suggestions of others in administering large doses of estrogens to such patients. We have recently carried 3 cases of premature rupture of the membranes for as long as twenty-seven, thirty-nine and forty-nine days.

DR. E. STEWART TAYLOR, Denver: Another generation of obstetricians would have been discussing, at a meeting such as this, methods to reduce maternal mortality. Physicians now agree that there is no reason for a woman to die in labor or as a result of pregnancy; however, there is a wide field open in the reduction of infant mortality. Since half the infant deaths in this country are associated with prematurity, this problem needs the attention of all physicians. It is important that physicians find a way to decide which premature cases need the most attention, and that is what my associates and I have attempted to do here. By giving special attention to abnormalities of the mother the large over-all fetal mortality of premature delivery and labor can be reduced.

DR. WILLIAM H. MASTERS, St. Louis: I agree with Dr. Kirschbaum. The entire point of the paper is that I do not believe general anesthetics, thiopental sodium or the barbiturates should be used in the conduction of premature labor. Dr. Kirschbaum's suggestion that scopolamine alone be used in the conduction of premature labor has not been universally accepted, nor am I aware of any comparable series in the literature where scopolamine was the only analgesic used in which the uncorrected premature mortality is no greater than that reported by Dr. Ross and myself. I disagree with Dr. Kirschbaum's statement that patients who have premature labor and deliveries under conduction anesthesia cannot be compared to similar patients whose labors are conducted without analgesia or anesthesia. In our admittedly small group the statistics of those patients who had premature labor without protection of any sort are much worse than the statistics of the conduction group. There was a 75 per cent infant mortality at the 1,500 Gm. weight level. We feel strongly that the premature infant needs protection during labor as well as at the time of delivery. I mentioned that the results obtained in our study were a matter of the participation of the house staff under residency supervision. The St. Louis Maternity Hospital did not have the six anesthesia teams that Dr. Kirschbaum suggests. It is simply a routine maternity hospital, and the results reported by Dr. Ross and me can easily be obtained by any comparable obstetric service and certainly could be improved by many. It is routine procedure that all conduction anesthesia is carried out by members of the house staff. In a series that now approaches 4,000 cases in which conduction anesthesia has been used, there have been no maternal deaths that in any way could be held the fault of the anesthesia methods. The statisticians who reviewed this paper estimated that as the result of the use of conduction anesthesia there were some 23 to 25 premature infants alive in and around the city of St. Louis who would not otherwise have survived.

DERMATOSES WHICH MAY HAVE AN ALLERGIC ETIOLOGIC BASIS

JAMES R. WEBSTER, M.D.
Chicago

This presentation is designed not to embody new ideas in cutaneous allergy but rather to give a brief clinical characterization of some of the dermatologic entities that experience has shown not infrequently arise as a result of altered reactivity. To decide whether or not a dermatosis may have an allergic basis, the first step is to make a dermatologic diagnosis if possible, and it is hoped that this presentation may be of help to the nondermatologist in this connection. The diagnosis will indicate whether allergy is a relatively certain, a possible or an unlikely etiologic factor, or need not be considered. It will also suggest the most probable form of allergy, if any, and the type of investigation required from this point of view.

In 1940 Sulzberger [1] listed a number of dermatoses, grouped as to likelihood of allergic causation as follows:

GROUP 1.—In many instances proved to be allergic.

1. Eczematous contact-type dermatitis
2. Certain infectious diseases such as:
   Tuberculoderms and ids
   Trichophytosis and trichophytids (dermatophytosis and dermatophytids)
   Syphilis and syphilids
3. Atopic dermatitis (including certain infantile eczemas and disseminate neurodermatitis (neurodermite) as well as certain prurigos)
4. Drug eruptions I (arsphenamine dermatitis; fixed eruptions; urticarial, morbilliform and scarlatiniform eruptions)
5. Serum sickness

GROUP 2.—Occasionally proved to be allergic.

1. Urticaria and angioneurotic edema
2. Purpuras (drugs, infections, etc.)
3. Drug eruptions II (acneform, lichenoid, furunculoid, erythema-nodosum-like, erythema-multiforme-like, etc.)
4. Erythema-nodosum-like and erythema-multiforme-like eruptions due to foods, foci of infection and causes other than drugs

GROUP 3.—Occasionally considered allergic on circumstantial evidence.

1. Erythema exudativum multiforme
2. Erythema nodosum
3. Herpes zoster
4. Dermatitis herpetiformis
5. Various purpuras
6. Many other skin diseases

(To this group might be added certain eczematoid eruptions of the hands as discussed by Flood and Perry [2])

Obviously time will not permit consideration of this entire list, and therefore only contact dermatitis, atopic dermatitis, the dermatophytids and certain drug eruptions will be taken up.

Contact Dermatitis (Eczematous Dermatitis; Dermatitis Venenata).—Contact dermatitis represents an acquired sensitization in which the shock tissue is the epidermis and the offending agent usually reaches the epidermis from the outside. This implies, in most instances, that dermatitis will not result immediately from the first contact with a substance, which differ-

Read before the Section on Miscellaneous Topics at the Ninety-Eighth Annual Sesion of the American Medical Association, Atlantic City, N. J., June 10, 1949.

1. Sulzberger, M. B.: Dermatologic Allergy, Springfield, Ill., Charles C Thomas, Publisher, 1940.
2. Flood, J. M., and Perry, D. J.: Role of Food Allergy in Eczematoid Dermatitis, Arch. Dermat. & Syph. 55: 493 (April) 1947.

entiates this phenomenon from inflammatory response to so-called primary irritants. It also follows that aside from instances due to applied medicaments, clothing and the like, dermatitis venenata is not often seen in infants and young children but is observed with increasing frequency as the patient encounters more potential sensitizers in later childhood and adult life.

The number and variety of substances capable of inducing eczematous sensitization is legion. Potential sensitizers are encountered in any walk of life; they may be solids, liquids or gases and commonly fall in one or more of the following groups: (1) locally applied medicaments; (2) cosmetics and other substances used in the toilet; (3) soaps, other detergents and other substances commonly encountered in the household or place of business; (4) clothing, other wearing apparel and substances carried in pockets; (5) various plants and plant products, especially lacquers, and (6) substances encountered in various occupations and industries or their products.[3]

From a diagnostic point of view contact dermatitis should be suspected when a dermatitis of eczematous character begins on a portion of the body where intimate contact with a sensitizer of some character is possible, whether or not the lesion remains localized in this vicinity. The location of onset will suggest causative possibilities in many cases, and charts dealing with this are available.[4] From this point a careful history will frequently indicate substances which are to be suspected. However, in many instances, especially in long-standing cases, the problem will not be so simple and much detective work involving the judicious and intelligent use of patch tests or elimination of possible factors from the environment and gradual replacement as advocated by Sutton [4] may be required.

Atopic Dermatitis (Diathetic Prurigo [Besnier]; Generalized Neurodermatitis [Neurodermite]; Eczematoid [Rost]).—Atopic dermatitis is a true antigen-antibody mechanism. The shock tissue would appear to be the vascular bed of the corium and the antigens, at least in the main protein in nature, are brought to the skin through the blood stream and derived from inhalants or ingestants. In many cases psychosomatic factors are added to those of altered reactivity, but evidences of the latter may be found in the majority of instances.

The disorder has a characteristic history and in late childhood and adult life a characteristic clinical appearance. It is commonly first noted in infancy, or at the latest in early childhood, when it is usually diagnosed as infantile eczema. Any part of the body may be involved, but even at this time the sites of predilection on face, neck, flexural surfaces of the extremities and upper trunk anteriorly and posteriorly may be noted. There is frequently a family history of eczema, hay fever or asthma. At times a definite relationship to articles of food recently added to the diet seems apparent.

The skin changes may vary from slight thickening and scaling with little itching to decided redness, edema, weeping, scaling and crusting with intense itching. If the eruption has continued for some time it presents a characteristic appearance even in relatively inactive phases by lichenification in the areas of predilection. In a definite majority of cases the disorder slackens in intensity between the ages of 2 years and 4 or 5 years; in many instances it disappears entirely. If this happens there may never be a recurrence, but in a significant number of cases there is recrudescence about the time of puberty. Once reestablished, the disease may last for many years with periods of relative remission and exacerbation. Eosinophilia of definite but low degree is usually found, and there may be associated asthma, hay fever or other allergic disorders.

In atopic dermatitis scratch and intradermal tests with food and inhalant extracts commonly result in a considerable number of immediate positive reactions, and circulating antibodies can often be demonstrated by the passive transfer (Prausnitz-Küstner) technic. However, the chief practical value of these tests may lie in helping to classify the disorder as an atopic process rather than in giving information of use in the management of the case. Dermatologists as a group prefer elimination and replacement of possible dietary and inhalant allergens, especially in children.

From a diagnostic point of view all the foregoing characteristics of atopic dermatitis are of interest. Differential diagnosis is probably most difficult in so-called infantile eczema, where the history and clinical appearance are frequently not clearcut. Seborrheic dermatitis and contact dermatitis due to soap, clothing and medicaments must be considered, and it will often be necessary to observe and study the patient over a considerable period of time before a reliable opinion may be given.

In older children or adults the diagnostic problem is usually easier, because the characteristic history of atopic dermatitis over a period of considerable time will be present and the characteristics of the eruption will be more apparent. Differential diagnosis in the older person must take into consideration contact dermatitis, seborrheic dermatitis, lichen simplex chronicus (localized neurodermatitis), so-called nummular eczema and the eczematization seen secondary to various parasitic infections, animal, bacterial and mycotic.

Dermatophytids.—According to Sulzberger [5] "an 'id' is a secondary manifestation appearing in a specifically altered (allergic) tissue and produced by micro-organisms emanating from a remote focus; and/or by allergenic products of such micro-organisms." Thus, a diagnosis of an id eruption postulates that in a person with a previously existing focus of infection a secondarily developed, specific, altered reactivity has been established; dissemination of the micro-organism and/or its products from the primary focus may then set up a more or less acute type of reaction in the sensitized skin, the clinical form of which will depend on many factors. The shock tissue in this case is in the corium and therefore intradermal tests are of value; but the response differs from that in urticaria and atopic dermatitis in that the reactions are of the delayed and usually papular variety.

I have chosen the so-called dermatophytids (trichophytids and epidermophytids) as an example for consideration, since they are relatively common conditions in which the primary focus as well as the secondary id manifestation is on the skin. Nearly twenty different forms of dermatophytid have been described, but I will discuss only two of the commoner forms, so-called vesicular and eczematous ids of the hands in association with a focus of dermatophytosis commonly on the feet.

Contrary to somewhat general belief, as has been shown by Hopkins [6] and others, a true dermatophytosis

3. Schwartz, L.; Tulipan, L., and Peck, S. M.: Occupational Diseases of the Skin, ed. 2, Philadelphia, Lea & Febiger, 1947.
4. Sutton, R. L., and Sutton, R. L., Jr.: Handbook of Diseases of the Skin, St. Louis, C. V. Mosby Company, 1949, p. 90.
5. Sulzberger,[1] p. 208.
6. Hopkins, J. G.: Personal communication to the author.

of vesicular character on the hands is a rather rare occurrence. However, vesicular and eczematous eruptions on the hands in association with a provable dermatophytosis of the feet are an exceedingly common occurrence, and one which offers a great deal of difficulty to the dermatologist from the point of view of differential diagnosis. There is a great temptation in cases in which one finds such a combination, and especially when there is a dermal sensitivity to trichophytin, to assume that the hand eruption is a pure dermatophytid. This is frequently not the case, since the eruption clinically may be closely simulated by dyshidrosis, contact dermatitis, various bacterids and in some cases, though more rarely, eczematous drug and food eruptions on the hands. In many such instances the hand eruption is in no way connected with the dermatophytosis of the feet; in others the dermatophytid is only part of the clinical picture on the hands. The diagnosis and management of these cases is therefore often extremely difficult. To have hope of being successful, the physician must take into account all the etiologic possibilities already mentioned and the multitudinous investigations suggested by them.

Probably the commonest clinical form of dermatophytid on the hands is that of recurrent crops of small pinhead to pinpoint-sized vesicles occurring in groups and having a special predilection for the interdigital surfaces of the fingers, but occurring also on the palms of the hands and infrequently on the dorsum. Often the appearance of vesicles closely follows an inflammatory exacerbation of the primary focus. Individual crops of vesicles may be absorbed without sequelae, but in some instances an eczematization will occur and in others secondary pyogenic infection will complicate the clinical picture. Another form of dermatophytid on the hands is so-called keratolysis exfoliativa, in which the vesicles are usually rather superficial and inconspicuous until the roof has been exfoliated and marginate spots are produced by the rim of loosened epidermis which remains at the periphery.

Clinically the differential diagnosis must include: (1) dyshidrosis, which is commonly associated with hyperhidrosis in persons with manifestations of anxiety; (2) vesicular contact dermatitis, which as a rule begins on the dorsa of the hands because of the relatively greater resistance of the skin on the palms; (3) infectious eczematoid dermatitis and nummular eczema occurring more commonly on the dorsa of the hands and fingers, and (4) the so-called pustular bacterid, which occurs on the palms and soles. This condition, which is also known as "recalcitrant pustular eruption of the palms and the soles" has been shown by Andrews and others to be traceable at times to a focus of pyogenic, usually staphylococcic, infection somewhere in the body.

Drug Eruptions.—The pathogenesis of drug eruptions and the manner in which allergy may play a part therein are much debated. In some of the more generalized types of eruptions it is believed that by conjugation with certain bodily proteins a drug may produce an antigen to which a specific antibody is formed and that the mechanism then closely parallels the process in certain urticarias or atopic dermatitis. In a few types of lesions the substance is thought to come to the epidermis through tissue fluid or via the secretions of skin glands and thus to induce an eczematous sensitization. In other types many authorities merely speak of an idiosyncrasy without other explanation. Occasionally an eruption will occur in a certain area or areas following the administration of a drug, will fade if the drug is not taken regularly but will recrudesce in the same area on readministration of the substance. Such phenomena are spoken of as fixed eruptions, and it is assumed that local sensitization has taken place.[7] With a few exceptions, such as a patch test in cases of eczematous dermatitis from internally administered drugs, skin tests used in allergy are not applicable in the investigation of drug eruptions. Of more value when a suspicion of a drug eruption arises is the elimination of the drug followed by healing of the eruption and its recurrence on readministration. In some cases chemical analysis of hair, nails, skin, urine or feces may be helpful, and bismuth deposits in muscle can be seen in roentgenograms.

Drug eruptions constitute a large number and variety of cutaneous lesions, from simple erythema to severe dermatitis. They include examples of all the elementary lesions of the skin and may simulate many other cutaneous disorders. A few are highly suggestive of certain drugs, such as generalized exfoliative dermatitis from arsenical compounds, fungating bromodermas or the eczematoid and hypertropic lichenoid syndrome seen in World War II from quinacrine hydrochloride. However, many different drugs may cause identical eruptions and many drugs may occasion more than one type of eruption. Generalized eruptions tend to be sudden in onset, symmetric, itchy and of a brighter color than disorders they simulate. Fixed drug eruptions are suggested by the foregoing classic history.

SUMMARY

Four examples of the many cutaneous disorders which may have an allergic etiologic basis have been briefly considered from a pathogenetic point of view to illustrate each of the four known types of cutaneous allergy and to indicate what type of investigation may be most helpful. Some of their diagnostic and differential diagnostic characteristics have been indicated.

122 South Michigan Avenue (3).

ABSTRACT OF DISCUSSION

Dr. Nelson Paul Anderson, Los Angeles: Dr. Webster has considered various dermatoses from the standpoint of allergy. This point of view has been the subject of papers, monographs and books which invariably take the position that since such ideas seem to be universally accepted, they should not be questioned. It may be worth while to consider two of the conditions emphasized by Dr. Webster. The first of these is contact dermatitis. The generally accepted view of its mode of development is that of an eruption produced by external agents acting as allergens. Many observations support this popular theory. These include the great specificity, the ease of experimental reproduction by the patch test, a period of refractoriness with a definite fixed incubation period and a fixed reaction time of twenty-four to forty-eight hours. There are also many observations which do not support this theory: 1. There has been failure to detect antibodies by any known tests. 2. Passive transfer is usually not successful. 3. Practically all substances causing contact dermatitis are nonantigenic or nonprotein in character: i. e., contact dermatitis is not ordinarily produced by true antigenic substances. 4. Subcutaneous, intravenous or intraperitoneal injection of simple chemicals is much less apt to produce epidermal sensitivity than the simple application of the same chemical in lesser amounts directly to the skin. 5. Experimentally, in order to sensitize animals to a simple chemical it is necessary to simultaneously inject large doses of tuberculin and/or a heterologous serum along with the simple chemical. 6. Spontaneous desensitization seldom

7. Ormsby, O. S., and Montgomery, H.: Diseases of the Skin, ed. 7, Philadelphia, Lea & Febiger, 1948.

occurs. 7. Therapeutic desensitization except in the case of plant excitants is rarely accomplished. 8. Recent work with the development of BAL (2,3-dimercaptopropanol) for arsenical and mercurial intoxications indicates that interference with an enzymatic system is responsible for the deleterious effect of these chemicals. Allergy as such is not involved. There is a great tendency to speak of various tissues as the "shock organs." Thus in contact dermatitis it is the epidermis. In the light of recent work by Peters and his co-workers on cutaneous vesicants it is necessary to probe deeper and to consider the physiology and biochemistry of the individual cell, and more particularly the enzymatic systems of the cells involved. The second group of conditions so often considered to be allergic in their causative mechanism are the drug eruptions. The recent and successful use of BAL in the treatment of acute arsenical eruptions as well as those due to gold, bismuth, and mercury compounds makes one feel that perhaps allergy as an etiologic mechanism has been overemphasized.

DR. LOUIS TUFT, Philadelphia: I get the impression from Dr. Webster's paper that skin tests are of little value in atopic dermatitis and that diets are preferable for diagnosis. That has not been my impression, nor that of most allergists. The reason for the widespread belief that cutaneous tests are of value is that foods have always been considered the predominant factor in atopic dermatitis. For a long time many physicians have emphasized the importance of inhalant factors; yet these have been almost completely neglected by dermatologists and even by many allergists. In a recent paper in the *Journal of Investigative Dermatology* I attempted to analyze the role of inhalants in atopic dermatitis. It was found, for example, that house dust is just as important a factor as foods and in many cases more important, especially in older children and adults. Danders of various sorts, wool and ragweed pollen —all are extremely significant in atopic dermatitis. Neglect of these inhalant excitants accounts, I believe, for many failures in the treatment of atopic dermatitis by dermatologists and explains the necessity of resorting to other explanations, such as the psychosomatic element. I am convinced that if one would look on atopic dermatitis as asthma of the skin and manage it in like manner, the results in atopic dermatitis would be much better. My own results in patients with atopic dermatitis are superior to those obtained in bronchial asthma, even though the same methods of diagnosis are used in the two conditions, it is important to remember that, as in bronchial asthma, patients with atopic dermatitis may manifest numerous positive skin reactions to certain food and inhalant allergens which seem to be of no clinical significance; nevertheless, unless the inhalant factors are given due consideration, the results of treatment may be worthless.

DR. JOHN L. FROMER, Boston: This discussion has a great deal of value for the allergist and the dermatologist. Fairly divergent opinions are held by Dr. Tuft and Dr. Anderson. When Dr. Anderson published his first paper in THE JOURNAL several years ago on methods of treating drug eruptions other than the simple allergic method, I considered that he had made a distinct contribution to the conception of some of these clinical phenomena. Contact dermatitis was long considered to be epidermoid in its origin or pathogenesis. It was thought to be an altered reactivity of the epidermis, the avascular epidermis that consists histologically of four layers. Atopic dermatitis was considered to be a sort of latent or subclinical urticaria that persisted and then resulted in epidermal changes. There grew up a concept that separated pathogenetically the epidermal sensitization of contact dermatitis and the vascular sensitization in atopic dermatitis. More recently there is evidence that perhaps these two concepts converge and that there is an epidermal-dermal sensitivity in both conditions. For a long time, it was believed that the two conditions could be differentiated by the dermatopathologist. Both present in some phase of their clinical manifestation hyperkeratosis, acanthosis, microscopic and gross vesiculation and spongiosis. In some cases of atopic dermatitis and of contact dermatosis there is vascular dilatation and an infiltration consisting of small and large round cells and, in many cases, eosinophils. The histologist therefore has great difficulty at times in differentiating atopic dermatitis from contact dermatitis and drug eruptions. Therefore one should continue to keep an open mind with respect to management of these disorders, particularly atopic dermatitis. For ten years I have seen many patients with these conditions, and, in contradistinction to Dr. Tuft's attitude, I would rather see an asthmatic patient than I would a patient with atopic dermatitis. In the treatment of atopic dermatitis associated with bronchial asthma, successful desensitization of the patient with inhalants, for example, improves the asthma or respiratory shock organ but the atopic dermatitis persists. The best management of atopic dermatitis lies between the two concepts presented today by the dermatologists and allergists. The allergist should know more about dermatology, and the dermatologist should know more about allergy.

DR. JAMES R. WEBSTER, Chicago: In attempting to cover this subject briefly one must be guilty of oversimplification and can hardly avoid making statements which may be considered controversial. This is particularly true as regards Dr. Anderson's remarks on drug eruptions. The practical value to the patient of intradermal and scratch tests in atopic dermatitis is obviously controversial as illustrated by Dr. Tuft's remarks. I must stand by my statement that dermatologists as a group prefer elimination and replacement of dietary and inhalant allergens as an investigative procedure in this dermatosis. I am of this opinion even after close cooperation on many such cases with well qualified allergists in Chicago. When it seems to me that the tests are indicated I refer the patients to an allergist, who can perform and interpret them much better than I can. The indiscriminate use of the term eczema causes much confusion. I sincerely believe that it would be preferable to eliminate the term from medical literature. In speaking of the two large groups of sensitization dermatitis I prefer for the sake of clarity to adhere strictly to the designations contact dermatitis and atopic dermatitis, to indicate that the pathogenetic mechanisms are for the most part entirely different.

Clinical Notes, Suggestions and New Instruments

MASKED MYASTHENIA GRAVIS

SYDNEY J. WEISMAN, M.D.
Los Angeles

The usual medical text infers that the diagnosis of myasthenia gravis is, as a rule, not difficult. In suspected cases the use of the therapeutic test as devised by Viets and Schwab[1] will usually leave no doubt as to the diagnosis. Similarly, the aggravation of the myasthenia by the administration of quinine or its derivatives is reported to make certain the diagnosis.[2]

The present case is reported because the diagnosis was unsuspected for a period of seven years, the main complaint being one not usually found in myasthenia gravis.

REPORT OF CASE

History.—A white woman aged 58 was first seen June 20, 1949. The presenting complaint was "palpitation" of seven years' duration; secondarily the patient complained of weakness and fatigue.

The family history was noncontributory; both parents had died of "heart disease," the father at 87 years of age and the mother at 79. Two siblings were alive and well. The patient had been married thirty-seven years previously but had been divorced for the past twenty-five years; there had been no pregnancies.

Surgical history included a tonsillectomy about twenty-five years previously and an appendectomy and right salpingectomy twelve years previously.

There was a history of "bloating" following the ingestion of freshly baked bread, pork and gravy. Pruritus ani had been

1. Viets, H. R., and Schwab, R. S.: Prostigmin in the Diagnosis of Myasthenia Gravis, New England J. Med. **213**: 1280, 1935. Viets, H. R., and Mitchell, R. S.: The Prostigmine Test in Myasthenia Gravis, ibid. **215**: 1064, 1936. Schwab, R. S., and Viets, H. R.: The Prostigmine Test in Myasthenia Gravis: Third Report, ibid. **219**: 226, 1938.

2. Harvey, A. M., and Whitehill, M. R.: Quinine As an Adjuvant to Prostigmin in the Diagnosis of Myasthenia Gravis: A Preliminary Report, Bull. Johns Hopkins Hosp. **61**: 216, 1937.

present for four years and had persisted despite much local therapy including roentgen irradiation. Menstruation had ceased completely in 1936; estrogenic substances had been administered until 1943.

In 1942, after the death of a friend, the patient noted the onset of palpitation. For the first nine weeks there was pronounced nervousness, insomnia and a loss of 25 pounds (11.3 Kg.). The palpitation occurred in attacks as often as five times a day and would last up to eight hours. The onset was sudden, with gradual disappearance in about one-half hour. A typical attack consisted of a sensation of a flutter in the left hypochondrium followed by sudden "skipping" and rapid cardiac rate. The patient had never counted the rate during an attack. Postprandial bloating, use of arms over head, housework and emotional distress served as precipitating factors. During the few weeks previous to her examination on June 20 the attacks had occasionally awakened her at night. There was no dyspnea. Severe exhaustion followed each episode of palpitation.

The weakness was moderate from 1942 to 1945; during 1945 it was less severe. However, during the past three years weakness became increasingly prominent. After walking about 150 yards (137 meters) she would feel as though her legs could not hold her up. One-half to one hour of housework caused complete exhaustion.

For the past two or three years there had also been episodes of moderately severe substernal burning pain with radiation through to the back. These occurred about every two to six months and were associated with dysphagia, though without regurgitation. Treatment had consisted of esophageal dilation.

The diagnosis had always been "extreme nervousness and esophageal spasm." Treatment had consisted of quinidine and sedation. The highest dosage of quinidine had been 15 grains (0.97 Gm.) per day, though usually no more than 9 grains (0.58 Gm.). The patient stated that she experienced no relief from quinidine; in fact, she remarked that quinidine seemed to increase the weakness. Pressure on the carotid sinus or on the eyeballs had failed to stop attacks of palpitation. Numerous metabolic rates had been determined and were all reported as normal. Roentgenologic examination of the gallbladder a year previously was reported as revealing a normally functioning organ.

Physical Examination.—The patient was well developed and well nourished. The voice seemed a bit weak and had a whining character. Her motions were for the most part slow.

Examination of the head revealed slight ptosis of the upper eyelids, more pronounced on the left. There was a sagging quality to the face. The neck and chest showed no abnormalities other than inversion of the right nipple. The apical cardiac rate was 62 per minute and was grossly irregular, with many premature contractions. The blood pressure measured 118 mm. of mercury systolic and 80 mm. diastolic. The only other positive observations were an atrophic vaginal mucosa, thickening and roughening of the perianal skin, a slight thoracic scoliosis to the left, minimal Heberden's nodes, and a moderate lipedema of the legs and ankles. Deep tendon reflexes and peripheral arterial pulsations were normal. Babinski great toe reflex and other abnormal reflexes were absent.

Fluoroscopic examination revealed the heart to be of normal size and configuration, the lungs to be clear and slight calcification to be present in the arch of the aorta. The electrocardiogram showed a few premature ventricular contractions, left axis deviation and a low T wave in lead 3; precordial leads (V leads) were normal.

Hemoglobin concentration measured 14.4 Gm. per hundred cubic centimeters. The red blood cells numbered 5,400,000 per cubic millimeter, the white cells 6,800 with a normal differential. Results of urinalysis were negative for sugar, albumin and cells. Reaction to serologic tests for syphilis were negative.

Treatment and Course.—Since it was not clear from the history that quinidine invariably increased symptoms, it was decided to treat the patient with quinidine and observe her closely. Digitalis was first administered and then gradually increasing doses of quinidine sulfate were given. After a dose of 15 grains of quinidine was reached, the patient began to complain of increased weakness. On July 2, 1949, the daily dose of quinidine being 24 grains (1.55 Gm.), the patient remarked that she was "almost an invalid, so weak I can't do anything." She could not comb her hair. The dose of quinidine was reduced to 18 grains (1.16 Gm.) per day. On July 6, 1949 she was confined to her house, unable to walk a few steps without palpitation; weakness was severe. On this date all medication was discontinued; the weakening effect of quinidine had been clearly demonstrated, and early and severe fatigue with any motion was obvious.

By July 9, 1949 the patient felt able to come to the office, and on that date the neostigmine test was performed. An injection containing 1.5 mg. of neostigmine methylsulfate and 0.6 mg. of atropine sulfate was given intramuscularly. The patient was not instructed as to any effects to be expected. Within ten minutes the patient remarked that she felt stronger. During ninety minutes of observation the usual side reactions occurred (twitching of muscles, increased intestinal activity and some slurring of speech); however, despite the unpleasantness of these effects, she insisted that strength and general sense of well-being were increasing. Objectively the patient was stronger, and the sagging character of the face decreased. The diagnostic numerical value of the test was 20, thus placing the reaction in the positive group.

On the evening of July 9, 1949 the patient began oral medication with neostigmine bromide, and this has continued to date. Three to five ¼ grain (15 mg.) tablets per day have sufficed to control symptoms. She feels better than she has since 1942 and each day enjoys engaging in activities which she had been unable to do for years without extreme exhaustion. Palpitation has ceased to be a major problem; there are only occasional abortive episodes. Her voice now is normal, and her general outlook has changed from one of mild depression to an attitude allowing her to enjoy life. The pruritus ani has become minimal, and the patient volunteered the information that quinidine had always increased the pruritus. The concomitant administration of atropine sulfate served to control most of the side effects of the neostigmine, effects which are most disturbing to the patient.

COMMENT

It is not to be inferred that in this patient the palpitation has been considered a manifestation of myasthenia gravis. The fact that the palpitation practically ceased after specific treatment was instituted would add credence to a suspicion that an interrelationship did exist. Conversely, one might believe that the palpitation was merely part of a functional overlay and that treatment with neostigmine caused the disappearance of the palpitation secondarily. The latter point of view is supported by Viets,[3] who has stated that in myasthenia gravis "heart action and other involuntary movements are not affected."

The severity of the case reported is obviously not great. Had it been, it is doubted that the patient would have survived the administration of quinidine over a period of seven years. The amount of neostigmine necessary to control the symptoms also attests to the relative mildness of the disease. However, the correctness of the diagnosis seems certain in view of the intensely severe reaction to the administration of quinidine and in view of the therapeutic response to neostigmine. It is of interest to note here that when the diagnosis was explained to the patient, she observed, "For years I've felt that quinidine would kill me if I took much more."

SUMMARY

A case of myasthenia gravis is reported. Its unusual feature is the prominence of the complaint of palpitation, prominent to the point that previous therapy always had been directed toward it. The patient ascribed the weakness primarily to the palpitation. The diagnosis was suspected on the observation of decidedly increased weakness following the administration of quinidine and on the history of dysphagia. The response to treatment with neostigmine was prompt and dramatic, and was accompanied with the almost complete cessation of palpitation.

6363 Wilshire Boulevard (36).

3. Viets, H. R.: Myasthenia Gravis, in Cecil, R. L.: A Textbook of Medicine, Philadelphia, W. B. Saunders Company, 1947, pp. 1536-1540.

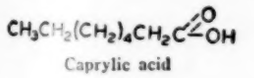
Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES

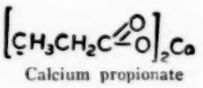
The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

AUSTIN SMITH, M.D., *Secretary.*

PROPIONATE-CAPRYLATE MIXTURES.—Sopronol Propionate-Caprylate Compounds (WYETH).—Preparations in which the formulation is varied with respect to both the ingredients and their concentrations according to the dosage form. The active ingredients are chosen from the following: calcium propionate, caprylic acid, propionic acid, sodium propionate, zinc caprylate and zinc propionate. Their structural formulas may be represented as follows:

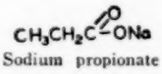


Caprylic acid

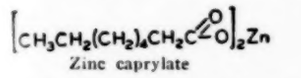


Calcium propionate

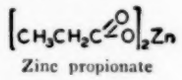
Propionic acid



Sodium propionate



Zinc caprylate



Zinc propionate

For description and standards for sodium propionate see the National Formulary.

Actions and Uses.—Propionate-caprylate compound is proposed for use against superficial fungous infections, especially dermatophytosis of the feet, hands and groin.

Dosage.—Cleanse the affected parts and apply morning and night.

Calcium Propionate.—The calcium salt of propionic acid.—$C_6H_{10}O_4Ca$.—M.W. 186.224.

Tests and Standards.—

Calcium propionate occurs as a white powder possessing not more than a faint odor of propionic acid. It is freely soluble in water and very slightly soluble in alcohol.

Carry out a flame test on the salt: a reddish-orange color indicates the presence of calcium.

Dissolve about 0.1 Gm. of calcium propionate in 10 cc. of water. Add ammonium oxalate T.S.: a white precipitate develops *(calcium)*.

Dissolve 1 Gm. of calcium propionate in 20 ml. of water. Add 3 drops of phenolphthalein T.S. and titrate with 0.1 *N* sulfuric acid: no more than 0.6 ml. is required to discharge the pink color.

Weigh, accurately, about 0.3 Gm. of calcium propionate. Dry at 105° C. for 1 hour: the loss in weight does not exceed 6.0 per cent.

Determine the calcium content as described in the monograph for Calcium Carbonate in U. S. P. XIII, p. 92. Each ml. of 0.1 *N* potassium permanganate is equivalent to 0.009311 Gm. of calcium propionate: the calcium propionate content is not less than 98 nor more than 102 per cent of the dried sample.

Caprylic Acid.—n-Octanoic acid.—$C_8H_{16}O_2$.—M.W. 144.21.

Caprylic acid occurs as a yellow to light amber, oily liquid with a characteristic goatlike odor. It is practically insoluble in water and very soluble in alcohol. Its refractive index at 25 C. is 1.4270. The specific gravity of caprylic acid at 25 C. is about 0.9110.

Shake 1 ml. of caprylic acid with 20 ml. of water, let stand and separate. Divide the aqueous phase into 2 portions. Acidify one portion with nitric acid and add 5 drops of silver nitrate T.S.: no turbidity appears (halides). To the other portion add 1 ml. of barium chloride T.S.: no turbidity develops (sulfates).

Weigh, accurately, about 1 Gm. of caprylic acid in a tared dish. Evaporate to dryness on a steam bath: the residue does not exceed 0.20 per cent of the original weight.

To 0.25 Gm. of caprylic acid in a separatory funnel, add 25 ml. of freshly prepared hydrogen sulfide T.S. and ammonia T.S. until the mixture is alkaline to litmus. Acidify with 2 ml. of hydrochloric acid and draw off the aqueous layer into a 50 ml. Nessler tube. Make this solution just alkaline to litmus with ammonia T.S., then add 5 ml. more. The greenish color produced, if any, should not exceed that of a control containing 10 ppm. of iron as ferroc ammonium sulfate (U. S. P. XIII, p. 730).

Weigh, accurately, about 0.2 Gm. of caprylic acid. Titrate the acid with 0.1 *N* potassium hydroxide. Each ml. of 0.1 *N* potassium hydroxide is equivalent to 0.014421 Gm. of caprylic acid: the caprylic acid content is not less than 97 nor more than 101 per cent of the dried material.

Propionic Acid.—Propanoic acid.—Methylacetic acid.—$C_3H_6O_2$.—M.W. 74.08.

Propionic acid occurs as a colorless liquid, possessing a characteristic odor and an acidic reaction. It is very soluble in water and in alcohol. The refractive index at 25 C. is about 1.3850. The specific gravity of propionic acid at 25 C. is 0.9924.

Add 2 ml. of water to 2 ml. of propionic acid. Add sufficient calcium chloride to saturate the solution: a layer of the acid appears on the surface of the solution.

Dissolve 1 ml. of propionic acid in 20 ml. of water. Add 5 drops of silver nitrate T.S.: no turbidity develops (halides).

Dissolve 1 ml. of propionic acid in 10 ml. of water and add 1 ml. of barium chloride T.S.: no turbidity develops (sulfates).

Mix 10 ml. of water with 2 ml. of propionic acid and add 0.1 ml. of potassium permanganate T.S. Allow to stand 2 hours: the color is not discharged.

Weigh, accurately, about 1 Gm. of propionic acid in a tared dish. Evaporate to dryness on the steam bath: the residue does not exceed 0.20 per cent of the sample as received.

Determine the heavy metals content according to the U. S. P. XIII, p. 657: the heavy metals content does not exceed 10 ppm.

Weigh, accurately, about 0.2 Gm. of propionic acid. Titrate the acid with 0.1 *N* potassium hydroxide. Each ml. of 0.1 *N* potassium hydroxide is equivalent to 0.007408 Gm. of propionic acid: the propionic acid content is not less than 99 nor more than 101 per cent of the dried material.

Zinc Caprylate.—The zinc salt of caprylic acid.—$C_{16}H_{30}O_4Zn$.—M.W. 351.784.

Zinc caprylate occurs as a very fine white powder. It is practically insoluble in water and in alcohol.

Dissolve about 0.1 Gm. of zinc caprylate in 10 ml. of water by adding 1 ml. of strong ammonia solution. Add sodium sulfide T.S.: a white flocculent precipitate develops (zinc).

Acidify about 0.1 Gm. of zinc caprylate with 10 ml. of sulfuric acid. Extract the solution with two 25 ml. portions of ether. Wash the ether solution with 3 successive 25 ml. portions of water. Evaporate the ether on a steam bath. The residue responds to the tests described in the monograph on Caprylic Acid—N.N.R.

Weigh, accurately, about 0.1 Gm. of zinc caprylate. Dry at 105 C. for 2 hours: the loss in weight does not exceed 3.0 per cent.

Boil 1.5 Gm. of zinc caprylate with 50 ml. of water and 10 ml. of hydrochloric acid. Filter the hot solution and wash the residue with about 50 ml. of hot water. Make the combined filtrate and washings alkaline with ammonia T.S., and add ammonium sulfide T.S. to precipitate the zinc completely. Dilute to 200 ml., mix and filter. Add 0.5 ml. of sulfuric acid to 100 ml. of the clear filtrate. Evaporate to dryness and ignite to constant weight in a muffle furnace at 500 to 700 C.: the weight of the residue does not exceed 7.5 mg.

Weigh, accurately, about 0.2 Gm. of zinc caprylate and transfer it to a 250 ml. beaker. Add 10 ml. of diluted sulfuric acid and heat the beaker on a steam bath until solution is effected. Dilute the solution to 100 ml. with water. Complete the analysis by directions given in the assay for Zinc Sulfate in U. S. P. XIII, p. 613, beginning with the statement, "Heat the solution to about 90° . . ." Each 0.1 Gm. of zinc oxide is equivalent to 0.4323 Gm. of zinc caprylate: the zinc caprylate content is not less than 95 nor more than 102 per cent of the dried sample.

Zinc Propionate.—The zinc salt of propionic acid.—$C_6H_{10}O_4Zn$.—M.W. 211.524.

Zinc propionate occurs as a very fine white powder. It is sparingly insoluble in water and in alcohol.

Dissolve about 0.1 Gm. of zinc propionate in 10 ml. of water by adding 1 ml. of strong ammonia solution. Add sodium sulfide T.S.: a white flocculent precipitate develops (zinc).

Acidify a small sample of the salt with sulfuric acid and warm: the odor of propionic acid is detected.

Weigh, accurately, about 0.1 Gm. of zinc propionate. Dry at 105 C. for 1 hour: the loss in weight does not exceed 4.0 per cent.

Boil 1.5 Gm. of zinc propionate with 50 ml. of water and 10 ml. of hydrochloric acid. Filter the hot solution and wash the filter paper with about 50 ml. of hot water. Make the combined filtrate and washings alkaline with ammonia T.S., and add ammonium sulfide T.S. to precipitate the zinc completely. Dilute to 200 ml., mix and filter. Add 0.5 ml. of sulfuric acid to 100 C. of the clear filtrate. Evaporate to dryness and ignite to constant weight in a furnace at 500 to 700 C.: the weight of the residue does not exceed 7.5 mg.

Weigh, accurately, about 0.3 Gm. of zinc propionate and suspend the powder in 10 ml. of distilled water in a 50 ml. distilling flask. Add 2.5 ml. of 85 per cent phosphoric acid, mix the contents of the flask and distil. Collect 10 ml. of distillate. Rinse the condenser with several 5 ml. portions of previously neutralized alcohol and add the rinsings to the distillate. Add sufficient neutralized alcohol to the distillate to assure complete solution. Titrate the solution with 0.1 *N* sodium hydroxide, using phenolphthalein T.S. as the indicator. Each ml. of 0.1 *N* sodium hydroxide is equivalent to 0.01058 Gm. of zinc propionate: the zinc propionate content is not less than 93 nor more than 102 per cent of the dried sample.

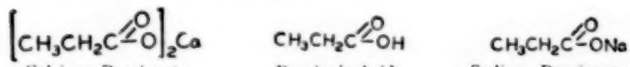
WYETH, INCORPORATED, PHILADELPHIA 3

Ointment Sopronol Propionate-Caprylates Compound: 30 Gm. tubes. A water-soluble ointment containing 12.3 per cent sodium propionate, 2.7 per cent propionic acid, 10 per cent sodium caprylate, 5 per cent zinc caprylate and 0.1 per cent dioctyl sodium sulfosuccinate.

Powder Sopronol Propionates-Caprylate Compound: 60 Gm. and 150 Gm. canisters. A dusting powder containing 15 per cent calcium propionate, 5 per cent zinc propionate, 5 per cent zinc caprylate and 0.25 per cent propionic acid in a talc base.

Solution Sopronol Propionate-Caprylate Compound: 60 cc. bottles. A dilute n-propyl alcohol solution containing 12.3 per cent sodium propionate, 2.7 per cent propionic acid, 10 per cent sodium caprylate, and 0.1 per cent dioctyl sodium sulfosuccinate.

PROPIONATE COMPOUND JELLY.—Propion (Wyeth).—A propionate-propionic acid mixture of calcium propionate 9.5 per cent, sodium propionate 9.5 per cent and propionic acid, 1 per cent marketed in the form of a water-miscible jelly. The structural formulas of the active ingredients may be represented as follows:



Calcium Propionate — Propionic Acid — Sodium Propionate

For description and standards for Sodium Propionate, see the National Formulary, and for Calcium Propionate and Propionic Acid see Tests and Standards under Propionate-Caprylate Mixtures which appears in this issue of The Journal.

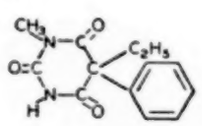
Actions and Uses.—Propionate compound is used in the form of a jelly for local application in the treatment of vulvovaginal moniliasis. Until more evidence becomes available, it is not recommended for other mycotic infections of the vulva or vagina, despite the fact that propionic acid compounds have been shown to be effective against a variety of fungous infections of the skin. It should be kept in mind that monilia are occasionally found in the vaginal secretions of apparently normal women and, when associated with Trichomonas infection, treatment of the latter sometimes suffices to clear up the symptoms. The relationship between these two organisms in vulvovaginitis is not yet completely understood.

Dosage.—Approximately 6 cc. of a jelly containing the propionate-propionic acid mixture of the aforementioned proportions is applied to the upper part of the vagina twice daily (morning and night) by means of an applicator. A single plain water douche is recommended before the first application, but douches are not permitted during treatment. A small amount of the jelly should be externally applied to the vulva. When there is considerable excoriation, the first two or three external applications should be made with the jelly diluted with an equal volume of water. Culture for monilia to determine cure may be taken two days after therapy has been discontinued. Vaginal applicators should not be used after the seventh month of pregnancy.

Wyeth, Incorporated, Philadelphia 3

Propion Gel. 95 Gm. tubes with vaginal applicator. A water-miscible jelly containing calcium propionate 9.5 per cent, sodium propionate 9.5 per cent and propionic acid 1.0 per cent in a base containing propylene glycol 10 per cent and bentonite 32 per cent.

MEPHOBARBITAL.—Mebaral (Winthrop-Stearns).—N-Methyl-5-ethyl-5-phenylbarbituric acid.—$C_{13}H_{14}N_2O_3$.—M. W. 246.26.—The structural formula for mephobarbital may be represented as follows:



Actions and Uses.—Mephobarbital produces the sedative effect characteristic of other members of the barbiturate series. Like phenobarbital, but unlike most other members of the series, mephobarbital also exerts a prominent anticonvulsant effect by selective action on the motor cortex, presumably due to the presence of the phenyl group. Clinical observations indicate that mephobarbital has comparatively mild hypnotic action so that it is better suited for use as a sedative than a soporific. Because its action in animals is not affected by the development of tolerance to other members of the barbiturate series, it is considered to have a different fate in the body than other derivatives of barbituric acid. It is excreted in the urine in traces only, yet exerts the prolonged action characteristic of phenobarbital and other long-acting compounds. The extent of its destruction by the liver has not been determined.

Mephobarbital is chiefly employed as an anticonvulsant for the treatment of grand mal and petit mal epilepsy, though there exists at present among workers in this field some difference of opinion regarding its degree of efficacy in these conditions. It is reported clinically to be somewhat more effective than phenobarbital in petit mal epilepsy. It may occasionally provide better control of both major and minor attacks than either phenobarbital or diphenylhydantoin sodium or when given alternately or in combination with either of those drugs. Mephobarbital is considered inferior to phenobarbital for the management of insane epileptics, but does control seizures in epileptic psychotic persons having only moderately advanced mental changes. It does not cure congenital mental defects or the mental deterioration often observed in epileptic persons. The drug may be used in conjunction with a ketogenic diet.

Mephobarbital is also useful as a sedative, especially in the treatment of agitated, depressed and anxiety states when minimal hypnotic action is desired. Mephobarbital produces side effects of drowsiness and gait disturbance similar to phenobarbital, but these appear to be less pronounced or persistent than with the latter drug. Such symptoms are indications for reduction of the dosage of the drug or its temporary withdrawal. As with other barbiturates, the drug should be used with caution in patients with impaired hepatic function.

Dosage.—Mephobarbital is administered orally in the form of tablets. In epilepsy, the average total daily dose for adults ranges from 0.4 Gm. to 0.6 Gm., though as little as 0.2 Gm. or as much as 0.8 Gm. may be required in some patients. Patients who have seizures principally at night who require not more than 0.4 Gm. daily may be given the entire dose at bedtime. For attacks during the day, half the daily dose required should be given during waking hours and half at night. Children under 5 years of age may be given a total daily dose of 0.03 Gm. to 0.06 Gm. and older children 0.06 Gm. to 0.3 Gm. Treatment should be started in all cases with a small initial dose and increased gradually over a period of four to five days until the optimum dosage is determined.

In combination with phenobarbital the dosage of each drug should be one-half the full dose as used alone. When the drug employed concurrently with diphenylhydantoin sodium, the dose of the latter should be reduced, but mephobarbital may be administered in full dosage as when given alone. Satisfactory results have been obtained with an average daily dose of 0.225 Gm. of diphenylhydantoin sodium plus 0.6 Gm. of mephobarbital.

As a sedative, mephobarbital may be administered in doses of from 0.03 Gm. to 0.06 Gm. three or four times daily, depending on the age and condition of the patient.

Tests and Standards.—

Physical Properties: Mephobarbital occurs as white, tasteless, odorless crystals which melt between 177 and 181 C. It is soluble in chloroform, slightly soluble in alcohol and ether and very slightly soluble in water. Mephobarbital dissolves in fixed alkali hydroxides and carbonates.

Identity Tests: Heat about 50 mg. of mephobarbital in a test tube with 0.2 Gm. of anhydrous sodium carbonate: a strip of moistened red litmus paper held in the mouth of the test tube burns blue.

Shake about 0.3 Gm. of mephobarbital with 5 ml. of 0.2 *N* sodium hydroxide and then filter. Add several drops of silver nitrate T.S. to 1 ml. of the solution: a white precipitate forms which dissolves readily in ammonium hydroxide. To another 1 ml. portion of the filtrate add several drops of mercuric chloride T.S. Remove the precipitate by centrifuging and wash it twice with water: the precipitate dissolves readily on the addition of ammonia T.S.

In a dry test tube mix 0.2 Gm. of mephobarbital, 0.5 Gm. of potassium nitrate and 2 ml. of sulfuric acid. Immerse the test tube in a bath of boiling water for 30 minutes, then cool the mixture and cautiously add 5 ml. of water. Make the solution distinctly alkaline with ammonia T.S. and heat until the evolution of gas ceases. Cool, filter and to 2 ml. of the filtrate add, without mixing, 2 drops of ammonium sulfide T.S.: a dark brown-red ring is formed *(distinction from barbital)*. Acidify a second 2 ml. portion of the filtrate with diluted sulfuric acid and recrystallize the precipitate from alcohol. It melts at about 220 C. (the phenobarbital derivative prepared in this manner melts at about 295 C.).

Purity Tests: Dry about 1 Gm. of mephobarbital, accurately weighed, at 105 C. for 4 hours: the loss in weight does not exceed 0.5 per cent.

Char about 0.5 Gm. of mephobarbital, accurately weighed, over a low flame. Cool, then add 1 ml. of sulfuric acid and continue ignition until no carbon remains: the residue does not exceed 0.1 per cent.

Assay: Transfer about 0.15 Gm. of mephobarbital, accurately weighed, to a semi-micro Kjeldahl flask and digest for 3 hours with 7 ml. of sulfuric acid, 2.7 Gm. of potassium sulfate and 0.3 Gm. of mercuric oxide. Dilute the clear solution to 15 ml. with water, make it alkaline with 40 per cent sodium hydroxide and then add 10 ml. of 40 per cent sodium thiosulfate. Distill the ammonia into 30 ml. of 4 per cent boric acid. Using a mixed indicator (1 part of methyl red T.S.: 5 parts of bromocresol green T.S.), titrate the liberated ammonia with 0.05 *N* sulfuric acid. Each ml. of 0.05 *N* sulfuric acid is equivalent to 0.0007004 Gm. of nitrogen: the nitrogen content is not less than 11.15 and not more than 11.50 per cent on a dry basis.

Transfer 0.25 Gm. of mephobarbital, accurately weighed, to a 150 ml. beaker and dissolve it by heating gently in 50 ml. of alcohol. Cool the solution, add 25 ml. of water and run an electrometric titration with 0.1 *N* sodium hydroxide using a glass electrode. Each ml. of 0.1 *N* sodium hydroxide is equivalent to 0.02463 Gm. of mephobarbital: the mephobarbital content is not less than 99 nor more than 101 per cent on a dry basis.

Mephobarbital Tablets: Crush 10 tablets and wash the powdered tablets with petroleum ether. Extract the washed material with chloroform in a Soxhlet extractor for 2 hours. Remove the chloroform by evaporation. The white residue melts between 177 and 181 C. and responds to the identification tests listed in the monograph on Mephobarbital.

Powder about 20 tablets and transfer the equivalent of about 0.3 Gm. of mephobarbital to a Soxhlet extractor thimble. Wash the sample with 15 ml. of petroleum ether and allow to drain. Transfer the thimble to the extraction apparatus and extract with chloroform for 2 hours. Quantitatively transfer the chloroform extract to a 150 ml. beaker and evaporate on a steam bath with the aid of a current of air. Add 50 ml.

of alcohol to the residue and heat until solution is complete. Add 25 ml. of water and titrate electrometrically with 0.1 *N* sodium hydroxide. Each ml. of 0.1 *N* sodium hydroxide is equivalent to 0.02463 Gm. of mephobarbital: the quantity of mephobarbital is not less than 94 nor more than 106 per cent of the claimed amount.

WINTHROP-STEARNS, INC., NEW YORK 13

Tablets Mebaral: 32 mg., 0.1 Gm. and 0.19 Gm.

U. S. patent 1,923,239; U. S. trademark 321,093.

CALCIUM LEVULINATE-N. F. (See New and Nonofficial Remedies 1949, p. 420).

The following dosage forms have been accepted:

CHICAGO PHARMACAL COMPANY, CHICAGO

Solution Calcium Levulinate: 10 cc. ampuls: A solution containing 0.1 Gm. of calcium levulinate in each cc.

CLIFFWOOD CHEMICAL CORPORATION, RICHMOND HILL 19, N. Y.

Powder Calcium Levulinate: Bulk.

CHORIONIC GONADOTROPIN (See New and Nonofficial Remedies 1949, p. 403).

The following dosage form has been accepted:

THE UPJOHN COMPANY, KALAMAZOO 99, MICH.

Powder Chorionic Gonadotropin: 10 cc. vials: A powdered preparation containing 5,000 I. U. of chorionic gonadotropin packaged with a 5 cc. ampul of injectable water as a diluent. Preserved with 5 mg. of chlorobutanol in each cc.

DIPHENHYDRAMINE HYDROCHLORIDE (See New and Nonofficial Remedies 1949, p. 23).

The following dosage form has been accepted:

PARKE, DAVIS & COMPANY, DETROIT 32

Solution Benadryl Hydrochloride: 10 cc. Steri-Vials: A solution containing 10 mg. of diphenhydramine hydrochloride in each cc.

U. S. patent 2,421,714; U. S. trademark 416,252.

ESTRADIOL-U. S. P. (See THE JOURNAL, Oct. 8, 1949, p. 390).

The following dosage forms have been accepted:

CHICAGO PHARMACAL COMPANY, CHICAGO

Solution Estradiol in Oil: 1 cc. ampuls and 30 cc. vials: A solution in sesame oil containing 0.14 mg. in each cc. and 0.28 mg. in each cc.

Suspension Estradiol with Benzyl Alcohol 4%: 30 cc. vials: A suspension containing 0.14 mg. in each cc. and 0.28 mg. in each cc.

ESTROGENIC SUBSTANCES (WATER INSOLUBLE) (See New and Nonofficial Remedies 1949, p. 367).

The following dosage form has been accepted:

THE CENTRAL PHARMACAL COMPANY, SEYMOUR, IND.

Solution Estronol in Oil: 10 cc. and 30 cc. vials: A solution in sesame oil containing 10,000 I. U. (1 mg.) in each cc. 10 cc. vials: A solution in sesame oil containing 20,000 I. U. (2 mg.) in each cc.

DICUMAROL (See New and Nonofficial Remedies 1949, p. 347).

The following dosage form has been accepted:

ELI LILLY & COMPANY, INDIANAPOLIS 6, IND.

Pulvules Dicumarol: 25 mg., 50 mg. and 100 mg.

DIGITOXIN-U. S. P. (See New and Nonofficial Remedies 1949, p. 267).

The following dosage form has been accepted:

ABBOTT LABORATORIES, NORTH CHICAGO, ILL.

Tablets Digitoxin: 0.15 mg.

CHOLINE DIHYDROGEN CITRATE (See New and Nonofficial Remedies, 1949, p. 425).

The following dosage form has been accepted:

CHEMO PURO MFG. CORP., LONG ISLAND CITY 1, N. Y.

Powder Choline Dihydrogen Citrate: 113.4 Gm. bottles.

ASCORBIC ACID-U. S. P. (See New and Nonofficial Remedies 1949, p. 560).

The following dosage form has been accepted:

THE VALE CHEMICAL CO., INC., ALLENTOWN, PA.

Tablets Ascorbic Acid: 25 mg. and 100 mg.

AMINOPHYLLINE-U. S. P. (See New and Nonofficial Remedies 1949, p. 322).

The following dosage forms have been accepted:

BREWER & COMPANY, INC., WORCESTER 4, MASS.

Solution Aminophylline: 20 cc. vials: A solution containing 25 mg. of aminophylline in each cc.

THE WM. S. MERRELL COMPANY, CINCINNATI 15

Solution Aminophylline with Benzyl Alcohol 2%: 2 cc. ampuls: A solution containing 25 mg. of aminophylline in each cc.

PHYSICIANS' DRUG & SUPPLY CO., PHILADELPHIA 6

Tablets Aminophylline: 0.1 Gm. and 0.2 Gm.

RIBOFLAVIN-U. S. P. (See New and Nonofficial Remedies 1949, p. 553).

The following dosage form has been accepted:

INTERNATIONAL VITAMIN DIVISION, IVES-CAMERON COMPANY, INC., NEW YORK 16

Tablets Riboflavin: 10 mg.

METRAZOL (See New and Nonofficial Remedies 1949, p. 278).

The following dosage form has been accepted:

BILHUBER-KNOLL CORP., ORANGE, N. J.

Powder Metrazol: 5 Gm., 25 Gm. and 100 Gm. bottles.

U. S. trademark 249,687.

OXIDIZED CELLULOSE (See New and Nonofficial Remedies 1949, p. 436).

The following dosage form has been accepted:

JOHNSON & JOHNSON, NEW BRUNSWICK, N. J.

Hemo-Pak Hemostatic Absorbable Uterine Gauze Packing (4 ply): In sealed tubes. 2 inches by 3 yards (5.08 by 374.32 cm.).

U. S. patents 1,939,235 and 2,232,990.

PENICILLIN FOR PARENTERAL USE FOR PROLONGED ACTION (See New and Nonofficial Remedies 1949, p. 153).

The following dosage form has been accepted:

CHAS. PFIZER & CO., INC., BROOKLYN 6

Crystalline Procaine Penicillin G (Micronized) in Oil: 1 cc. and 10 cc. vials: A suspension in sesame oil with 2 per cent aluminum monostearate containing 300,000 units in each cc.

SULFAMERAZINE-U. S. P. (See New and Nonofficial Remedies 1949, p. 138).

The following dosage form has been accepted:

MARVIN R. THOMPSON, INC., STAMFORD, CONN.

Suspension Sulfamerazine with Sodium Lactate: 473 cc. bottles: A suspension containing 0.1 Gm. of sulfamerazine and 0.3 Gm. of sodium lactate in each cc.

U. S. patent 2,460,437.

PENICILLIN FOR ORAL ADMINISTRATION (See New and Nonofficial Remedies 1949, p. 156).

The following dosage form has been accepted:

ABBOTT LABORATORIES, NORTH CHICAGO, ILL.

Soluble Tablets Crystalline Potassium Penicillin G: 100,000 units.

IODOCHLOROHYDROXYQUINOLINE-N. F. (See New and Nonofficial Remedies 1949, p. 202).

The following dosage form has been accepted:

CHEMO PURO MFG. CORP., LONG ISLAND CITY 1, N. Y.

Powder Iodochlorohydroxyquinoline: 28.3 Gm. bottles.

MERBROMIN-N. F. (See New and Nonofficial Remedies 1949, p. 102).

The following dosage form has been accepted:

CHEMO PURO MFG. CORP., LONG ISLAND CITY 1, N. Y.

Crystals Merbromin: 150 Gm. bottles.

METHAMPHETAMINE HYDROCHLORIDE (See New and Nonofficial Remedies 1949, p. 227).

The following dosage form has been accepted:

THE VALE CHEMICAL CO., INC., ALLENTOWN, PA.

Tablets Dexoval Hydrochloride: 2.5 mg. and 5 mg.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

535 NORTH DEARBORN STREET - - - CHICAGO 10, ILL.

Edited by
MORRIS FISHBEIN, M.D.
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Subscription price - - - - Twelve dollars per annum in advance

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SATURDAY, NOVEMBER 26, 1949

FELLOWSHIP IN THE AMERICAN MEDICAL ASSOCIATION

According to the constitution and by-laws of the American Medical Association, active members of the Association who are in good standing may apply for Fellowship in the Scientific Assembly. Applications may be forwarded to the Secretary of the Association and are examined by the Judicial Council if the applicants are to be considered as Member or Service Fellows, or in some instances as Affiliate Fellows. Applications for Associate Fellowship, some Affiliate Fellowships and Honorary Fellowships are acted on by the House of Delegates. Member Fellows are from the active members of the Association; Service Fellows are from commissioned medical officers of the United States Army, the United States Navy and the United States Public Health Service and from the permanent medical officers of the Veterans Administration; Associate Fellows are from those who have been Fellows for not less than fifteen years and who are not less than 65 years of age, or who are members of their component societies and their constituent association but who are relieved from the payment of the dues or fees imposed on active members; Affiliate Fellows are from five classes, and Honorary Fellows are composed of physicians of foreign countries who have risen to preeminence in the profession of medicine. At the December meeting of the American Medical Association the House of Delegates will be asked to consider revision of the by-laws to permit qualified commissioned medical officers of the United States Air Force to apply for Service Fellowship.

Member Fellows pay annual dues of $12. Included in the dues is subscription to THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION or any special scientific journal published by the Association. Service Fellows and Associate Fellows do not pay dues but do not receive any publication of the Association except by personal subscription.

Members in good standing of their component county and constituent state or territorial medical associations become members of the American Medical Association when their names are reported officially to the Association by the proper officials of the constituent associations. Such members must take the initiative and make application for Fellowship if they wish to become Fellows. Apparently many members of the American Medical Association are unaware of this, as records at the Association headquarters reveal they subscribe individually to THE JOURNAL or a special journal without applying for Fellowship. Perhaps some erroneously believe that membership and subscription provides Fellowship. More is needed, although it is a simple act to apply for Fellowship.

Fellowship in the Association provides advantages for the Fellow and the Association. A Fellow may be chosen as a delegate, elected as an officer in the Association, present papers at the Scientific Assembly and otherwise engage actively in Association affairs. Only Fellows can be officers of the Association and members of its House of Delegates. In fact, a member must have been a Fellow for two years prior to the session at which he represents his constituent state or territorial medical association in the House of Delegates. A Fellow is designated in the records of the Association by the symbol ⊕ which appears after his name. This symbol indicates the bearer's interest in participating actively in the work of the American Medical Association. Of the more than 144,300 physicians who are members of the American Medical Association, approximately 81,000 are Fellows. Lack of understanding probably is responsible for many members not being Fellows. If a member or Fellow is in doubt concerning his status, he may inquire of the Secretary of the Association, 535 North Dearborn St., Chicago 10.

THE NEPHROTIC SYNDROME

The genesis of the so-called nephrotic syndrome has not thus far been elucidated. It is convenient from a clinical point of view to regard it as an independent entity. The syndrome is by some believed to be renal in origin, either a disease entity per se or a phase in chronic glomerulonephritis, while others believe it to be of extrarenal origin, caused by some disturbance of the protein metabolism. The syndrome, as pointed out by Bradley and Tyson,[1] presents certain uniform and consistent features, such as gross edema, hypoproteinemia, hypercholesteremia, lipidemia and heavy proteinuria in the absence of congestive heart failure.

The plasma protein composition is greatly altered in the nephrotic syndrome. There is a significant reduction in albumin, while the globulin content may rise, especially in amyloidosis, may remain unchanged or

1. Bradley, S. E., and Tyson, C. J.: The "Nephrotic Syndrome," New England J. Med. **238**: 223 (Feb. 12) 1948. Bradley, S. E., and Tyson, C. J.: "Nephrotic Syndrome" (Concluded), New England J. Med. **238**: 260 (Feb. 19) 1948.

may fall. The urinary proteins are probably derived from plasma by a filtration mechanism. The obvious site of this filtration is the glomerulus. Proteinuria in the nephrotic syndrome is attributed to defective glomerular activity, even though the glomeruli show little or no gross alteration. The urine is highly concentrated and contains cellular elements of casts of various kinds in great numbers. Renal functional and anatomic abnormalities invariably are demonstrable in the nephrotic syndrome. They are, however, never sufficiently severe to suggest extensive damage and loss of tissue. These processes are characteristically reversible. The return of the plasma protein concentration toward normal, as the urinary protein loss decreases, indicates that proteinuria is most important in the pathogenesis of hypoproteinemia. There is no evidence that protein synthesis is stimulated by additional protein intake.

The most distressing and disabling manifestation of the nephrotic syndrome is the edema. Lowered plasma protein concentration is most likely the responsible factor in the formation of the edema. Some believe that renal dysfunction in the disposal of water and electrolytes is chiefly concerned. It is also held that a specific fault in the renal excretion of sodium is the important factor. The alleviation of the nephrotic syndrome during various infectious diseases has frequently been observed. Janeway and his associates and Blumberg and Cassady noted a pronounced diuresis in children with chronic glomerulonephritis who contracted measles. Seegal and Wertheim [2] state, however, that in some patients the occurrence of an infectious disease in the course of the nephrotic syndrome may prove detrimental, inducing hematuria and further accumulation of edema. Patients with the nephrotic syndrome are lacking in resistance and usually die of an intercurrent infection.

One of the most puzzling manifestations of the nephrotic syndrome is the disturbance of the fat metabolism that gives rise to hypercholesteremia and hyperlipemia. The fat content of diet appears to have little influence on the plasma composition. The cause of lipidemia in nephrosis is obscure. The subclinical forms of the nephrotic syndrome are best detected, according to Seegal and Wertheim, by the determination of the serum albumin and serum cholesterol values. An abnormally low serum albumin level is evidence of the presence of the nephrotic phase. Hypercholesteremia is a confirmatory finding. The development and the disappearance of the nephrotic phase is not characterized by precise anatomic and physiologic changes in the kidney.

Lange and his associates [3] believe the nephrotic stage to be a generalized disease of the small blood vessels with a tremendous increase in capillary permeability. The edema formation, in their opinion, is due to this fact and not primarily to the lowered colloid osmotic pressure.

The diuretic effect of the administration of salt-poor human serum albumin produced usually only a transitory diuretic effect and did not influence the course of the syndrome. Administration of gum acacia is not effective and probably not altogether safe. Seegal and Wertheim conclude that the most useful agent in the control of edema of renal origin is the dietary restriction of sodium chloride.

The fundamental cause of this syndrome remains in doubt. Treatment also is veiled with uncertainty. However, with the development of new technics for studying tissues and cellular changes it may be possible to learn more of this interesting and distressing medical disease. Until then, unfortunately, management of the patient with a nephrotic syndrome will present a perplexing clinical problem.

2. Seegal, D., and Wertheim, A. R.: Recent Advances in Our Knowledge Concerning the Nephrotic Syndrome, Bull. New York Acad. Med. **25**: 605 (Oct.) 1949.

3. Lange, K.; Weiner, D., and Boyd, L. J.: Nephrosis: New Concepts of Functional Pathology and Therapy of the Nephrotic Stage, Section on Exhibits, J. A. M. A. **134**: 62 (May 3) 1947.

INTER-ASSOCIATION COMMITTEE ON HEALTH

On November 14 the formation of an Inter-Association Committee on Health was announced.

Six national associations, the American Dental Association, the American Hospital Association, the American Medical Association, the American Nurses Association, the American Public Health Association and the American Public Welfare Association have joined forces to study and attempt to find solutions to the many problems connected with health in the United States. These are the associations most vitally concerned with the delivery of medical care in all its aspects.

The first meeting was held July 20, 1949 and was called by the American Medical Association. The Hospital, Medical, Public Health and Public Welfare associations were represented. At this meeting it was decided to issue invitations to the Dental and Nurses associations to join in this project. All six associations took part in the second meeting on August 20, when it was decided that a more or less permanent committee should be formed to serve as a forum for discussion of mutual problems.

Principles of organization have been adopted by the Committee. The Board of Trustees of the American Medical Association has approved these principles, and it is expected that the other organizations will do so soon. According to these "Principles" the Committee is organized for these objectives: "To study and discuss various phases of health care in the interest of improving the health of the nation. It shall serve as a medium of exchange of information on the programs and interests of the participating organizations to the end that, so far as possible, a common understanding

may be reached toward a solution of health problems of mutual interest encountered in these programs. The committee shall perform such functions and carry on such activities contributing to the major objectives as may be satisfactory to the appointing organizations."

Each organization is to be represented by three officers or members of policy-making bodies. Staff members may attend in a nonvoting capacity. Preliminary discussions have been held on a wide variety of topics, and it is expected that announcements will be made from time to time as to recommendations or conclusions agreed on by the committee.

Each of the six organizations may well be proud of the part it is playing in this new project to improve the nation's health.

Current Comment

REGISTRATION AT THE WASHINGTON MEETING

The Clinical Session of the American Medical Association at Washington (December 6-9) may be attended by Member or Service Fellows; Associate, Affiliate or Honorary Fellows, invited guests, qualified physicians who apply for fellowship, members of the Association, medical students of approved medical schools who are certified to the Secretary of the Association by their respective deans, and interns and residents who are graduates of approved medical schools and who are certified to the Secretary of the Association by the superintendents of their respective hospitals. Those who have not used the Advance Registration coupon, which has appeared at frequent intervals in THE JOURNAL, may register at the Registration Bureau in the National Guard Armory, which will be open at 8:30 a. m. on Tuesday, December 6.

CARE BOOK PROGRAM

The need of technical books for scientific and cultural reconstruction in some foreign countries is urgent. Demand for such material far exceeds present publishing facilities in these countries, and gifts of money are needed to overcome this problem. CARE, through its CARE Book Program, is attempting to obtain books and periodicals for universities and other cultural institutions. A contribution coupon may be found on advertising page 24, of this issue of THE JOURNAL. Donors of $10 or more may specify, if they wish, the country, type of institution and category of books; however, books cannot be sent directly to individuals through this program. This is not a program to seek used books, and books should not be sent by contributors. The Board of Trustees of the American Medical Association has voted to support efforts to obtain appropriate books and periodicals for distribution through CARE. Checks and money orders may be sent to CARE Book Program, CARE, 20 Broad Street, New York 5.

THE USE OF TELEVISION

Medical societies should give serious thought to television as a medium for presenting programs on health education. Television is still a young industry; even in many of the larger cities stations are just being erected. The developmental period inevitably will be attended by presentation of a considerable amount of inferior audiovisual material. Thus if the medical society can offer such stations interesting, lively subjects with appropriate visual properties, it may be sure the offer will be accepted gladly. Ventures into this field should not be taken lightly, but it is unnecessary to adopt an attitude that it is territory in which only specialists or professional persons operate. This is an industry that is operating with little or no profit, and emphasis on contribution of time as a public service arouses little if any enthusiasm. The stations want good shows that cost as little as possible. The medical profession is in an unique position in this respect, for there are literally countless subjects that lend themselves readily and completely to television presentation. This matter is considered extensively in a television handbook issued by the Bureau of Health Education of the American Medical Association. Suggestions contained therein can serve as the starting point for the development of ideas that can be put to practical use to meet local conditions. Appreciation of what the local medical society has to offer will be heightened if the show is delivered as a "package." The script should be prepared in detail, although it will not ultimately be read as such; exhibits should be itemized and their use indicated, and satisfactory participants should be obtained. By careful study local and state medical societies can increase their public service and can gain another opportunity to become better known by the general population.

ANTIBIOTICS AND BLOOD COAGULATION

In 1945 Moldavsky [1] found that the blood of patients receiving penicillin injections clotted more rapidly than normally. A similar phenomenon was afterward reported for both penicillin and streptomycin when given by mouth by Macht [2] of Sinai Hospital, Baltimore. Macht and Farkas [3] have found that 200 mg. of aureomycin caused the normal coagulation time of cats to be reduced from 11 to 4 minutes within 75 minutes after oral administration. In rabbits an oral dose of 100 mg. shortened the time to 1.5 minutes. In human beings the normal time of 8 minutes was shortened to 5 minutes after administration of 1 capsule containing 250 mg. of aureomycin and further reduced to 3.5 minutes after administration of a second capsule. They believe that the definite shortening of the clotting time at the height of antibiotic therapy is of practical clinical interest.

1. Moldavsky, L. F.; Hasselbrook, W. B., and Cateno, G. D.: Science **102**: 38, 1945.
2. Macht, D. I.: Science **105**: 213, 1947.
3. Macht, D. I., and Farkas, R.: Science **110**: 305, 1949.

ORGANIZATION SECTION

Official Notes

CLINICAL SESSION, WASHINGTON, D. C., DEC. 6-9, 1949

Several features of the Clinical Session at Washington, D. C., will be of special interest to the physician in general practice.

Premier Showing of Cancer Film

The American Cancer Society, with the cooperation of the National Cancer Institute, will have a premier showing of a new motion picture, entitled "Breast Cancer, the Problem of Early Diagnosis," in the South American Room, Hotel Statler, Washington, D. C., 2:30 p. m., on Monday, December 5. All physicians are invited. The film will also be shown daily at 1 p. m. in the Motion Picture Theater, Room 9, in the Scientific Exhibit at the National Guard Armory during the meeting.

Fracture Demonstrations

The Special Exhibit Committee on Fractures has announced that demonstrations will begin Tuesday morning, December 6, for the benefit of physicians who register early. Drs. Kellogg Speed, Chicago, chairman; Gordon M. Morrison, Boston, and Frederick A. Jostes, St. Louis, will be assisted by the following demonstrators:

John J. Byrne, Boston
Milton C. Cobey, Washington, D. C.
Edward J. Coughlin Jr., Williamstown, Mass.
John J. Crowley, Lynn, Mass.
John L. Doherty, Boston
S. S. Freedman, Lowell, Mass.
Nicholas J. Giannestras, Cincinnati
Morris E. Goldman, Lewiston, Me.
E. D. Hagerty, Manchester, N. H.
Harry B. Hall, Minneapolis
Frank M. Hand, Washington, D. C.
Eugene A. Hamilton, Chicago
T. Wiley Hodges, Washington, D. C.
Earle E. Hussey, Fall River, Mass.
Elwood K. Jones, Bridgeport, Conn.
John J. Kelleher, Lawrence, Mass.
Wm. M. Lanigan, Medford, Mass.
Allen Lloyd, Washington, D. C.
John J. Milroy, Waukegan, Ill.
Edgar M. Neptune, Syracuse, N. Y.
Julius S. Neviaser, Washington, D. C.
Cecil E. Newell, Chattanooga, Tenn.
Austin Rohrbaugh, Chevy Chase, Md.
William J. Tobin, Washington, D. C.
Kenneth R. Weston, Philadelphia

The Washington representative of the Committee is Leonard T. Peterson.

RECORDED TRANSCRIPTIONS FOR STATE SOCIETIES

The Bureau of Health Education has discontinued for 1950 its network radio series and is directing its resources toward the provision of special service for state and local societies. This will permit an increase in the number of recorded transcriptions for state societies during the year.

On December 1 a series of thirteen programs entitled "The Drugs You Use" will be ready for release. The programs in this series will include the following titles and speakers:

1. Use and Misuse of Drugs.....Dr. Austin Smith
2. Germ Chasers.....Dr. E. M. K. Geiling
3. Pain Killers and Sleep Producers.....Dr. Carl Pfeiffer
4. Laxatives and Indigestion.....Dr. H. D. Kautz
5. Asthma and Hay Fever.....Dr. Harry L. Huber
6. Cosmetics.....Mrs. Veronica L. Conley and Dr. Howard T. Behrman
7. Skin Remedies.....Dr. Howard T. Behrman
8. Heart, Kidney and Blood Pressure Ailments....Dr. Arthur C. DeGraff
9. Hormones and Fat Reducers.....Dr. Sidney C. Werner
10. Safeguarding New Drugs.....Dr. Walton Van Winkle Jr.
11. Quackery.....Mr. Oliver Field
12. Your Prescription and Your Druggist.....Dr. R. Blackwell Smith
13. A Look to the Future.....Dr. E. M. K. Geiling

On January 1 three additional series will be available. The first of these, under the title "Your Child Goes to School," features discussions by school health authorities on such problems as boxing in the schools, interscholastic activities in junior high school, vending machines and school health examinations. The second of these will feature Jim Ameche in a dramatic narrative presentation of the work of great American physicians. The third series will include the thirteen choice programs from Ben Park's documentary series "It's Your Life." This series, broadcast in Chicago, has won every conceivable radio award, and several have been created especially for it. It will be presented by the American Medical Association under a new title, probably "The Living Proof." This series is built on tape-recorded interviews with actual patients who have benefited by modern medical treatment. It is an extraordinary emotional and compelling presentation.

Other recordings will be made available during 1950.

Washington Letter

(From a Special Correspondent)

Nov. 21, 1949.

Medical Care of Dependents Subject of Controversy

A proposal by the Budget Bureau, which is part of the President's executive offices, that the armed forces discontinue medical and hospital care to servicemen's dependents aroused a storm of controversy. Such action, said Army Surgeon General Raymond W. Bliss, would work irreparable damage to morale, besides jeopardizing the residency and internship training programs of military hospitals.

The proposal, which is certain to meet with strong opposition in Congress, made by Budget Director Frank Pace to Secretary of Defense Johnson was that both inpatient and outpatient services be terminated by Jan. 1, 1950. Dr. Richard L. Meiling, medical services director for the Department of Defense, took no positive stand for or against the plan but advised that no change be made before July 1, 1950, to protect residents now in training at Army and Navy hospitals. Addressing the Society of Medical Consultants in World War II on November 14, Dr. Meiling intimated that abolition of dependent care was under consideration and that economic retrenchment might force a reduction in the medical departments' postgraduate training and research programs.

Veterans Home Town Medical Care

Veterans Administration has issued a reminder to beneficiaries seeking home town medical care for service-connected disabilities that they must first be certified by the nearest Veterans Administration regional office.

"An increasing number of claims for reimbursement have come to the Veterans Administration Board of Appeals from veterans who failed to obtain Veterans Administration approval before going to their home town doctor for treatment," said an official statement. "Except in those cases where it was found that emergency treatment for a service-connected disability had been necessary, the claims have been disallowed."

Increase in Red Cross Blood Collections

Washington headquarters of the National Blood Program, American Red Cross, reports that collections have begun to increase since the midsummer slump. In October they totaled 43,012 pints, 10 per cent above the September figure. Seventeen of the 30 regional centers reported new highs for October, and all but seven experienced increases in collections over the previous month. Up to November 1, collections since the program's launching aggregated 426,204 pints of blood, donated by 353,538 persons. Distribution was among approximately 1,400 hospitals in 35 states. Included among the recipients were 80 independent clinics.

Sir Philip Manson-Bahr Lectures on Filariasis

Civilian and military physicians of Greater Washington will hear a lecture on "Problems of Filariasis" December 1 at the National Institutes of Health by Sir Philip Manson-Bahr. A son-in-law of the late Sir Patrick Manson and an authority on tropical diseases in his own right, Sir Philip is passing through this country on his way to Fiji, where he will join his son in studies of filariasis.

GOVERNMENT SERVICES

ARMY

170 BOARD MEN IN ARMY MEDICAL CORPS

Among the 1,457 regular officers in the Army Medical Corps as of September 30, there were 160, about 11 per cent, who were certified by American specialty boards, according to figures released by the Surgeon General's office. In addition, there were 10 specialists among Medical Corps Reserve officers on active duty on that date. Included in the total Regular Army figures are 27 retired officers recalled to duty.

Represented in the ranks of the uniformed physicians and surgeons are 18 specialties. The Army has 47 officers certified in internal medicine, 20 in radiology and 10 or more in clinical pathology, general surgery, pathologic anatomy and psychiatry. There are also one or more specialists in anesthesia, dermatology and syphilology, obstetrics and gynecology, neurology, ophthalmology, orthopedic surgery, otolaryngology, pediatrics, physical medicine, plastic surgery, preventive medicine and public health, thoracic surgery and urology. The Army is seeking additional board men in these fields, as well as in allergy, cardiology, gastroenterology and pulmonary diseases. Under the Graduate Professional Training Program, the Army has residents in both military and civilian teaching hospitals, in training for board examinations in almost all the specialties mentioned. Also, under the Civilian Consultants Program, many civilian specialists assist in the teaching of the younger officers, both at home and abroad, and otherwise contribute their skills toward the accomplishment of the mission of the Army Medical Department.

ARMY PIONEERS OF PREVENTIVE MEDICINE

Nine Army physicians who left legacies of health to the nation in the field of preventive medicine were featured by the Surgeon General's Office in an exhibit at the recent annual convention of the American Public Health Association in New York City. The exhibit, which was entitled "Pioneers in Preventive Medicine, U. S. Army," was built around the achievements of the following medical officers:

John Shaw Billings (1838-1913), prime mover in the expansion of the Army Medical Library, now one of the largest compilations of medical works in the world. Among his accomplishments was the expansion of the Index Medicus from one small pamphlet to many large volumes.

James Tilton (1745-1822), Surgeon General from 1813 to 1815, who established early standards of ventilation and hospital construction. Tilton provided the precedent for present day four bed cubicles.

Carl R. Darnall (1867-1941), inventor of the first machine for applying gaseous chlorine to water, one of the great contributions in the field of preventive medicine.

Benjamin Waterhouse (1754-1846), who introduced smallpox vaccination into America in 1800 (THE JOURNAL, Nov. 12, 1949, page 794).

William A. Hammond (1829-1900), Surgeon General from 1862 to 1864, who projected the plan for the Army Medical School, America's oldest school in preventive medicine, now called the Army Medical Department Research and Graduate School. He is probably best known as the founder of the Army Medical Museum, now the largest in the United States.

William C. Gorgas (1854-1920), Surgeon General from 1913 to 1918, whose sanitary control made possible the construction of the Panama Canal.

George M. Sternberg (1838-1915), Surgeon General from 1893 to 1902, widely known as the "father of American Bacteriology" and author of the first American textbook on that subject.

Walter Reed (1851-1902), leader of the team that verified Finlay's theory on the mode of transmission of yellow fever and renowned conqueror of that disease.

Frederick Fuller Russell (born 1870), who introduced typhoid vaccination into the U. S. Army. Russell is the only one of the "nine pioneers" who is still living.

ADVANCED TRAINING

The following additional medical officers have been accepted for advanced training by civilian institutions:

Lieut. Col. Isaiah A. Wiles, University of Oregon Medical School, Portland, gastroenterology.
Capt. Walter E. Horn, University of Pennsylvania Graduate School of Nursing, Philadelphia, dermatology.
Capt. Walter A. Schoen Jr., Medical College of Virginia, Richmond, obstetrics and gynecology.
Capt. George E. Ollag, City Hospital, Akron, Ohio, surgery.
Capt. Melton P. Meek, New York Medical College, New York, pediatrics.
Capt. Ralph E. Campbell, Medical College of Virginia, Richmond, psychiatry.
Capt. Donald J. Joseph, St. Louis University School of Medicine, otolaryngology.
Capt. Harry W. McCurdy, Coisinger Memorial Hospital, Danville, Pa., otolaryngology.
First Lieut. John M. Harter, Johns Hopkins Hospital, Baltimore, psychiatry.
First Lieut. Francis T. Rafferty, Colorado Psychopathic Hospital, Denver, psychiatry.
First Lieut. Norman B. Yourish, University of Pennsylvania Graduate School of Medicine, Philadelphia, ophthalmology.
First Lieut. Philip E. Duffy, Hospital of the University of Pennsylvania, Philadelphia, neurology.
First Lieut. Alexander H. Beaton, Strong Memorial Hospital, Rochester, N. Y., otolaryngology.
First Lieut. George R. Nicholson, City Hospital, Akron, Ohio, pathology.
First Lieut. John A. Hightower, University Hospital, Baltimore, internal medicine.
First Lieut. Thomas G. Baskin, John Sealy Hospital, Galveston, Texas, neuropsychiatry.
First Lieut. Philip R. Dodge, City Hospital, Boston, neurology.
First Lieut. Bruce T. Forsyth, Barnes Hospital, St. Louis, medicine.
First Lieut. Ralph V. Gieseiman, Barnes Hospital, St. Louis, medicine.
First Lieut. Thomas T. Glasscock, Grace-New Haven Community Hospital, New Haven, Conn., psychiatry.
First Lieut. Wood B. Herren, King County Hospital, Seattle, medicine.
First Lieut. Bruce R. Marshall, St. Luke's Hospital, Cleveland, otorhinolaryngology.
First Lieut. Robert F. Ranson, Charity Hospital, New Orleans, pathology.
First Lieut. Maurice S. Rawlings, George Washington Hospital, Washington, D. C., medicine.
First Lieut. William L. Stone, George Washington University Hospital, Washington, D. C., medicine.
First Lieut. Lawrence L. Washburn Jr., Louisville General Hospital, Kentucky, internal medicine.
First Lieut. Louis E. Young, Grace-New Haven Community Hospital, New Haven, Conn., surgery.

CONFERENCE ON INDUSTRIAL HYGIENE

Col. Wesley C. Cox (MC), consultant to the Surgeon General in occupational health and hygiene, left November 23 for Argentina for the first Inter-American Conference on Industrial Medicine, to be held in Buenos Aires December 1 to 15 under the auspices of the Argentine Ministry of Labor, the Ministry of Health, the University of Argentina and the Permanent International Commission on Workers' Medicine. Colonel Cox will speak on "Industrial Medicine in the Department of the Army." Representatives of eighteen Western Hemisphere nations will attend; among the European experts present will be Dr. Thomas E. A. Stowell, Royal Scientific Society, London; Dr. Luggi Carozzi, University of Geneva; Dr. Rene Fabre, University of Paris; Dr. Enrico Carlo Vigliani, University of Milan; Dr. Alfonso de la Fuente y Chaos, University of Madrid, and Dr. Jarolslav Teisingir, Charles University, Prague.

PERSONAL

Col. Byron L. Steger (MC) has been assigned as assistant chief, Education and Training Division, Surgeon General's Office, replacing Col. Lloyd E. Gould (MC), who transferred to the Air Force School of Aviation Medicine, Randolph Field, Texas.

PHYSICIANS SEPARATED FROM SERVICE

ARMY MEDICAL CORPS OFFICERS RECOMMENDED FOR/OR RELIEVED FROM ACTIVE DUTY

California

Antipa, Milton A.....San Francisco
Arklin, Virgil P.....Hollywood
Bazzanella, Robert P.....San Francisco
Becker, Donald L.....Grass Valley
Clark, Robert E.....Monterey Park
Cole, Leon R.....Beverly Hills
Dalby, Robert B.....Los Angeles
Dreyer, Clifton.....Hollywood
Gleason, Richard L.....Hemet
Grossman, Moses.....San Francisco
Hjelte, George S.....Los Angeles
Kennedy, Robert A.....Ojai
Krag, Daniel O.....Los Gatos
Kring, Harvey W.....San Francisco
Lewis, Richard D.....Long Beach
Maass, Lyman.....Antioch
Mitchell, Daniel A. Jr.....Los Angeles
Nelson, Donald G.....Los Angeles
Padel, Eugene G.....Alameda
Poytress, Richard W.....San Jose
Rhea, Thomas E.....Riverside
Smith, Edward J. .....San Francisco
Thee, Earl J. Jr.....Los Angeles
Vanina, Robert C.....Sacramento
Wallerstein, Ralph O.....San Francisco
Wass, Warren A.....Upland
Welsh, Joseph E. Jr.....Santa Cruz
Wheeler, Stanley D.....Loma Linda
Whipple, Gerald H.....Berkeley
Willey, Roger W.....Santa Rosa
Wright, Stanley W.....Berkeley

Illinois

Arnolde, Herbert A.....Chicago
Atherton, Leon G.....Peoria
Buczynski, Charles C.....Chicago
Dobrow, Bernard.....Chicago
Echt, Raymond J.....Chicago
Froom, Jack.....Chicago
Hall, Richard E.....Willow Springs
Hirsch, Frederick J.....Oak Park
Levine, Robert S.....Chicago
Lichtman, William F.....Chicago
Lindahl, Elbert L.....Crystal Lake
MacCarthy, John D. Jr.....La Grange
McFarlane, Peter J.....Hartford
McLaughlin, John R.....Chatham
Mizera, Thaddeus F.....Chicago
Moore, Thomas S.....Chicago
Moshein, Jack.....Pontiac
O'Reilly, Clarence J.....Chicago
Schweinfurth, James P.....Pekin
Segal, Peter B.....Chicago
Stephens, George S.....Chicago
Trippel, Otto H.....Chicago
Wilson, John A.....Pekin

Minnesota

Aufderheide, Arthur C.....New Ulm
Engstrom, Denton P.....Minneapolis
Linner, Paul W.....Minneapolis
McCool, Robert F.....St. Paul
Mahaffy, John H.....Minneapolis
Mandel, Sheldon L.....Minneapolis
Midthune, Andreen S.....Borup
Person, Waldemar N.....Delano
Peteler, Jennings L.....Glen Lake
Shragg, Robert I.....Minneapolis
Sperling, Sydney C.....Minneapolis
Thomas, John V.....Duluth
Wood, Newell E.....St. Paul

New York

Alder, Daniel H.....Brooklyn
Alfano, Joseph E.....Buffalo
Andaloro, William S.....Buffalo
Bell, Samuel D. Jr.....New York
Bennett, William W.....Brooklyn
Berman, Morris.....Brooklyn
Blumberg, Arnold G.....Brooklyn
Brainard, Scott C.....New York

New York—Continued

Cyran, Stanley J. Jr.....Buffalo
Dembrow, Victor David.....Brooklyn
Dewolfe, Phillip W.....Garden City
Flieschmann, Charles A.....Brooklyn
Forgrave, Edward G.....Tonawanda
Gittelson, Stanley B.....Brooklyn
Glesmann, John B.....Rome
Gordon, Joseph G.....Jamaica
Green, Marvin.....Brooklyn
Greenberg, Irving M.....Hempstead
Harris, Irwin L.....Brooklyn
Hippert, Gordon J.....Lackawanna
Howard, Chester S.....Buffalo
Jacobs, Jack.....New York
Jones, Robcliff V. Jr.....Bronxville
Kessler, Edward.....Brooklyn
Kiley, John E.....Troy
Kinley, Charles H.....Breesport
Klauber, Leo D.....Staten Island
Kleiner, David.....Brooklyn
Lacey, Thomas H.....Rochester
Lange, William A.....Flushing
Lazarus, Victor C.....Buffalo
Lieberman, Jerrold S.....New York
Liegner, Leonard M.....Jamaica
Loree, Harry E. Jr.....New York
Madell, Warren.....Bronx
Mazursky, Milton M.....New York
Mazza, Ralph J.....Ithaca
Muehsam, Gerald E.....New York
Nassberg, Seymour.....New York
Paley, George E.....Brooklyn
Reynolds, Francis X.....New York
Rubin, Stanley M.....Yonkers
Rumore, Thomas P.....Brooklyn
Ruskin, Edwin R.....New Rochelle
Sageman, Clifford B.....Saranac Lake
Seibel, David I.....New York
Shorr, Jay.....Brooklyn
Soffer, Alfred.....Rochester
Steele, Edward F.....Elmira
Tague, William F.....Syracuse
Vogt, Frank C.....Brooklyn
Warshaw, Lesley M.....Laurelton
Webb, Charles R.....Buffalo
West, William K.....Baldwinsville
Williams, Myron E. Jr.....Batavia
Wroblewski, Felix.....Brooklyn
Zaino, Edward C.....Islip

Ohio

Anderson, James M.....Columbus
Badley, Dana N.....Cleveland
Benua, Richard S.....Columbus
Black, Joseph M.....Shaker Heights
Blydenburgh, George D.....Delaware
Brown, Michael.....Cincinnati
Chess, Walter K.....New Concord
Gaughan, John J.....Leetonia
Greenlees, John P.....Marietta
Hannum, John N. Jr.....Lakewood
Harvey, Robert A.....Cleveland
Hoeflinger, George R.....Columbus
Holden, Willard Mc.....Akron
Janovsky, Ray C.....Cleveland
Levine, Albert H.....Cleveland
McMillen, Robert S.....Shadyside
Meckstroth, Norman A...New Knoxville
Moffet, Dale V.....Dayton
Murphy, Robert J.....Columbus
Platt, Daniel E.....Cleveland
Pritchard, Jack A.....Cleveland
Raub, Roy R.....Cushing
Rimelspach, John J.....Columbus
Scheuerle, Roland F.....Cincinnati
Schroer, George J.....Coldwater
Shneider, Julian S.....Cincinnati
Snyder, Donald A.....Clyde
Suder, Garfield L.....Marietta
Swisher, Marion R.....Eaton
Weisman, Philip A.....Dayton
Wetmore, Stanley M.....Youngstown

Oklahoma

Hale, Arthur E.....Alva
Hyroop, Gilbert L.....Oklahoma City
Kelso, Kip G.....Oklahoma City
Mathews, Charles R.....Oklahoma City
Miller, Ross H.....Ada
Moore, Walter M.....Muskogee
Lowry, David C.....Oklahoma City
Pitts, James B. Jr.....Oklahoma City
Price, Richard D.....El Reno

Oregon

Du Vall, Clyde H. Jr.....Portland
Holloway, James W.....Portland
Langley, Ivan I.....Portland
Markee, Franklin K.....Canby
Oberg, Stanton G.....Portland
Sanders, Donald D.....Dallas
Selling, Ben.....Portland
Smith, Lendon H. Jr.....Portland
Struthers, Robert A.....Portland
Stupfel, James F.....McMinnville
Vancil, Gordon Q.....Portland
Vogel, Marion P.....Portland
Westman, Charles W.....Portland

Pennsylvania

Barbanti, Amedeo A.....Philadelphia
Barr, Samuel, S.....Lancaster
Beck, William C. Jr.....Sewickley
Borkovic, George W.....Vanport
Brooks, Gerald M.....Meadville
Caddy, James A.....Johnstown
Carabasi, Ralph A.....Cynwyd
Catanzaro, Charles.....Norristown
Cochran, Robert A.....Glenside
Cohen, Norman.....Pittsburgh
Cramer, Bernard.....Philadelphia
Dereamer, John W.....Morrisville
Detrick, Bill D.....Philadelphia
Dolphin, Joseph M.....Wilkes-Barre
Eckberg, John J.....North Irwin
Fidler, Harry E.....Hooversville
Fisher, Herbert.....Philadelphia
Friedmann, Jacob B.....Philadelphia
Gailey, Herman A. Jr.....York
Goren, Norman J.....Philadelphia
Gosztonyi, R. E. Jr.....Northampton
Gouldthorpe, Dunstan D.....Erie
Groverman, Lester J.....Philadelphia
Hampe, Warren W. Jr.....Philadelphia
Hause, Clark D. W.....Philadelphia
Hofammann, Karl E. Jr.....Altoona
Holman, Theodore L.....Millerstown
Jacobs, George J.....Pittsburgh
Jennings, David T.....Pittsburgh
Johns, Donald C.....Donora
Kane, William M.....Wilkes-Barre
Keller, John M.....Philadelphia
Kleckner, Martin S. Jr.....Allentown
Knowles, Charles A.....West Pittston
Krevsky, Seymour.....Allentown
Krisanda, Joseph B.....Jessup
Kujda, Dominic A.....Dickson City
Lacock, Robert R.....Pittsburgh
Langfeld, Stephen B.....Melrose Park
McHenry, Thomas.....Ambridge
McMillan, Thomas M. III...Philadelphia
Makler, Paul T.....Philadelphia
Mann, Richard H.....Millersville
Matteucci, Walter V.....Philadelphia
Meyer, Harold.....Philadelphia
Minick, Russell C.....New Castle
Natali, Daniel E.....Elizabeth
Neumayer, Francis.....Philadelphia
Palin, William E.....Burgettstown
Palmer, Frank C.....Johnstown
Patrick, Thomas E.....Scranton
Perkins, Benjamin S.....Conshohocken
Pierce, Frank F.....Danville

PHYSICIANS SEPARATED FROM SERVICE

Pennsylvania—Continued

Propst, Harry D.....Scranton
Prutzman, Lewis D.....West Chester
Ranii, Carmello A.....Blawnox
Reed, Wilbur G.....Johnstown
Reitz, Melvin L.....Allentown
Rhoads, John M.....Philadelphia
Rider, Paul W.....Colonial Park
Ruht, Joseph C.....Shenandoah
Shore, George R.....East Brady
Sieber, Paul R. Jr.....Pittsburgh
Sokoloff, Louis.....Philadelphia
Sproch, Thomas M.....Latrobe
Thiers, George F.....Pittsburgh
Valin, Robert J.....Erie
Walters, William H....New Philadelphia
Weaver, James D.....Erie
Wissinger, John P.....Philadelphia
Wojnar, Victor S.....Ardmore
Yevitz, Michael G.....Scranton

Rhode Island

Banfield, Wm. G. Jr.....Saunderstown
Boyd, James F. Jr.....Providence
Caruolo, Joseph E.....Providence
Forsythe, Thomas.....Pawtucket
Johnson, Linwood H. Jr.....Westerly

South Carolina

Burgess, Rembert O.....Spartanburg
Dennis, Leroy B. Jr.....Florence
Goggans, Walter H.....Newberry
Rivers, Walker P. Jr.....Aiken
Ruff, John D.....Ridgeway
Simpson, James L.....Richburg
Watson, Paul E. Jr.....Enoree
Wheeler, Paul C.....Columbia

South Dakota

Aspaas, Paul K.....Garretson
Nelson, Robert E.....Pierre
Rivin, Arthur U.....Scotland

Tennessee

Ackerman, Robert F.....Memphis
Barr, James R.....Selmer
Brooks, Eugene M.....Nashville
Clayton, George W. Jr.....Memphis
Crotzer, Malcolm C.....Clarksville
Edmondson, Paul J.....Memphis
Evans, Paul H.....Knoxville
Klein, Victor H. Jr.....Knoxville

Texas

Bozzell, James D.....Weatherford
Haag, Edmund L. Jr.....Dallas
Jarvis, Garth L.....Austin
Longino, Stephen B. Jr...Sulphur Springs
Lumpkin, Forrest E. Jr.....Terrell
McKenzie, Charles E.....Miami
Nicolaou, George T.....San Antonio
Robins, Eli.....Rosenberg
Steed, Thurmon M. Jr.....Atlanta
Verheyden, Floyd H.....Dallas
Williams, Bryan Jr.....Marshall

Utah

Benson, Byron N.....Ogden
Wilcox, Milton F.....Ogden

Virginia

King, Casper Sidney.....Lynchburg
Painter, William G.....Big Stone Gap
Perkins, Horace E.....Richmond
Vaughan, David D.....Richmond

Washington

Buckley, John D.....Seattle
Cole, Menford D.....Seattle
Garrard, Willis D.....Kennewick
Heath, Sherburne W. Jr.....Olympia
Langenbach, Raymond J.....Spokane
Murphy, John C.....Spokane
Musser, Richard E.....Tacoma
Nelson, Donald W.....Edmonds
Nelson, Sidney W.....Lyden
Olson, Arthur J.....Malo
Weed, James W.....Longview
West, Thomas R.....Tacoma
Zahn, Albert C.....Tacoma

West Virginia

Bane, Earle M.....Charles Town
Boone, Reece R. Jr.....Smithers
Gibson, Robert E.....East Charleston
Knox, Kenneth R.....Parkersburg

Wisconsin

Brousseau, Edward R.....Wauwatosa
Bullock, Albert A. Jr.....Milwaukee
Chudwin, Manny, N.....Madison
Fetherston, John P. Jr.....Milwaukee
Hunsader, Hugo N.....Madison
Kaufman, Kiesl K.....Milwaukee
Lund, George W. .....Cumberland
Millenbah, Jack D.....Gleason
Segnitz, Richard H.....Milwaukee
Stannard, Gilbert H. Jr.....Sheboyan
Talbert, Myron J.....Waupaca
Torkildson, Gus C.....Milwaukee
West, Robert C.....Madison

NAVY

COURSE FOR RESERVE OFFICERS

The sixth course for Naval Reserve medical officers in the medical aspects of special weapons and radioactive isotopes was held the week beginning November 14. Rear Admiral W. S. Parsons, member of the Weapons Systems Evaluation Group, Office of the Secretary of Defense, spoke November 15 on "Naval Problems of Atomic Warfare" and Dr. Shields Warren, professor of pathology, Harvard University Medical School, on "The Fundamental Biology of Ionizing Radiation." On November 17, Col. James P. Cooney, U. S. Army, addressed the group on "Medical Problems of Radiologic Safety." Colonel Cooney was at Bikini in 1946 and Eniwetok in 1947. R. E. Lapp, Ph.D., spoke on "Fission and Ionizing Radiation." Dr. Lapp was executive director on atomic energy and advisor to the Office of Civil Defense Planning, Atomic Energy Commission. November 18, Livingston Satterthwaite, deputy director of the Office of British Commonwealth and Northern European Affairs, spoke on "The British Commonwealth as a World Force."

FIRST ACTIVE DUTY

The following reserve medical officers have been nominated for their first active duty:

Comdr. Rees Morgan, Roanoke, Va.
Comdr. William A. Tyler, Canton, N. Y.
Lieut. Comdr. Richard S. Clark, Redwood City, Calif.
Lieut. Comdr. Ralph C. Warne, Doheny Park, Calif.
Lieut. (jg) Edward T. Bonar, Columbus, Ohio.
Lieut. (jg) Bruce J. Ebbels, Rockville Centre, N. Y.
Lieut. (jg) David P. Hall, Chickamauga, Ga.
Lieut. (jg) David E. Loveman, Gadsden, Ala.
Lieut. (jg) David S. Masland, Carlisle, Pa.
Lieut. (jg) Arpad L. Masley, Madison, Wis.
Lieut. (jg) Guy P. Sharpe, Tchula, Miss.
Lieut. (jg) John P. Allan, Chapel Hill, N. C.
Lieut. (jg) Anthony J. Guida, San Carlos, Calif.
Lieut. (jg) Robert R. Nardone, Newton, Mass
Lieut. (jg) Thomas F. Dillon, Raquette Lake, N. Y.
Lieut. (jg) George O. Hilgermann, Minneapolis.
Lieut. (jg) John Hinman, Woodside, Calif.
Lieut. (jg) Jack C. Lindsey, Mount Victory, Ohio.
Lieut. (jg) Richard L. Shriner, Cassopolis, Mich.

DUTY UNDER INSTRUCTION

The following regular Navy medical officers have been nominated for duty under instruction in the Navy's Graduate Medical Training Program:

Comdr. Wendell A. Butcher, to a residency in psychiatry, Naval Hospital, Bethesda, Md.

Comdr. Francis G. Soule Jr., to a residency in internal medicine, Naval Hospital San Diego, Calif.

Comdr. Harold F. Thompson, to a residency in psychiatry, Naval Hospital, Philadelphia.

Comdr. Dana D. Goldthwaite, to a residency training in orthopedic surgery, Naval Hospital, Philadelphia.

Lieut. Comdr. James A. Turner, to a fellowship in pathology, Wayne University College of Medicine, Detroit.

Lieut. Robert M. Campbell, to a residency in general surgery, Naval Hospital, Long Beach, Calif.

Lieut. Harry A. Weiss, to a residency in internal medicine, Naval Hospital, San Diego, Calif.

Lieut. Elmer F. Lowry Jr., to a residency training in psychiatry, Naval Hospital, Bethesda, Md.

Lieut. Romulus L. May, to a fellowship in surgery, Georgetown University Hospital, Washington, D. C.

Lieut. John F. McCabe, to a residency training in pathology, Naval Hospital, Oakland, Calif.

Lieut. (jg) Marvin S. Allen, to a residency in pediatrics, Naval Hospital, Philadelphia.

Lieut (jg) Jack E. Byrd, to a residency in obstetrics and gynecology, Naval Hospital, Porthmouth, Va.

Lieut. (jg) Richard F. Dobbins, to the Naval Submarine School, New London, Conn.

Lieut (jg) Donald W. Edwards, to a residency in pediatrics, Naval Hospital, Philadelphia.

Lieut. (jg) Albin Gedarovich, to a residency in pediatrics, Naval Hospital, Bethesda, Md.

Lieut. (jg) Pierce P. Newman Jr., to the Naval Submarine School, New London, Conn.

Lieut. (jg) Paul Hart, to a residency training in internal medicine, Naval Hospital, St. Albans, L. I., N. Y.

Lieut. (jg) Robert O. Peckinpaugh, to a residency training in internal medicine, Naval Hospital, Long Beach, Calif.

AIR FORCE

COURSES OPEN TO OFFICERS

Two courses for medical officers are being offered at the School of Aviation Medicine, Randolph Air Force Base, Texas. These are the flight surgeon's course and the aviation medical examiner's course. Only regular Air Force officers are eligible to attend the flight surgeon's course, which is of ten months' duration and is a comprehensive presentation of aviation medicine. The course includes about 60 to 80 hours of flying instruction, flight indoctrination in new type aircraft, involving all weather flying, high altitude flying, jet flights and glide controlled landings, so that students may gain a practical understanding of the physical and mental problems which confront flying personnel.

The aviation medical examiner's course is open to any medical officer on active duty whose period of service extends a year beyond completion of the course. The course, which is of 11 weeks' duration, is devoted to air crew maintenance and care of flyers, preventive and global medicine, physiology, neuropsychiatry and ophthalmology.

Graduates may, on fulfilling certain requirements, apply for a flight surgeon's certificate at the completion of a year's duty in aviation medicine.

CIVILIAN CONSULTANTS AT AIR FORCE HOSPITALS

One hundred and forty-three civilian physicians, representing eighteen fields of medicine and surgery, have been chosen as expert consultants to 38 hospitals of the U. S. Air Force Medical Service, according to an announcement by Major Gen. Malcolm C. Grow, the Surgeon General. Consultants may be called on by the commanding officer of the station hospital for professional advice or appropriate professional assistance. They are regarded as members of the professional staff of the commanding officer of the hospital and are available for care and consultation of patients in the hospital and outpatient clinic.

PUBLIC HEALTH SERVICE

NEW MEMBERS OF THE NATIONAL HEART COUNCIL

The following new members of the National Advisory Heart Council were announced October 23: Louis W. Jones, Fayetteville, Ark., president of the University of Arkansas; Dr. H. M. Marvin, New Haven, Conn., associate clinical professor of medicine, and Mr. Erwin Oreck, Duluth, Minn., president of Oreck's Incorporated and director of the Duluth Fatherless Boys' Association. The new appointees will succeed the following members, whose terms are expiring: Mr. James S. Adams, New York City; Mr. Ernst Mahler, Neenah, Wis., and Dr. Irvine H. Page, Cleveland.

FEDERAL AID FOR RESEARCH

Public Health Service grants of $907,212 to aid laboratory and clinical research were made by the National Cancer Institute to nonfederal hospitals and universities in 21 states and the District of Columbia after recommendation by the National Advisory Cancer Council and approved by the Surgeon General. With these funds several institutions will investigate the relation of cancer and hormones. Under a grant to Memorial Hospital, New York City, Drs. R. W. Rawson and H. J. Tagnon will study the role of the human liver in reducing the activity of estrogen, a female sex hormone which has been proved capable of inciting cancer in mice. A tracer study of cortisone, a hormonal product of the adrenal glands, will be supported through a grant to the St. Louis University School of Medicine. By tagging cortisone with radioactive carbon, the investigator, Dr. E. A. Doisy, will trace the drug through the bodies of laboratory animals. Previous studies by Dr. Doisy, a Nobel Prize winner, have shown that less than 10 per cent of administered cortisone is excreted in the urine, but the remainder has not been accounted for.

BRUCELLOSIS

In a panel discussion on brucellosis October 18 at the Communicable Disease Center, Atlanta, Ga., Dr. James H. Steele, chief, Veterinary Public Health Division of the Center, said that brucellosis is now recognized as a major public health problem in the United States. Reported cases have increased from 60 in 1927 to more than 6,000 in 1947, he said. It is estimated, however, that the actual number of cases exceeds 40,000 a year. About 5 per cent of all the cattle in the country are believed to be infected. The economic loss to the livestock industry from this disease "is conservatively estimated," Dr. Steele concluded, at more than $100,000,000 yearly.

MISCELLANEOUS

PROFESSOR BALE APPOINTED RADIOBIOLOGIST

The Atomic Energy Commission announces that William F. Bale, professor of radiation biology at the University of Rochester Medical School, has been appointed to the staff of the U. S. Atomic Energy Commission as radiobiologist of the Division of Biology and Medicine. Dr. Bale will be responsible for the biologic and health aspects of the commission's waste disposal program. While associated with the commission, he will be on leave from the University of Rochester, where he was head of the Division of Radiology and Biophysics of the Atomic Energy Project.

COURSES IN RADIOISOTOPE TECHNIC

Vacancies still remain for the winter series of three courses in radioisotope technics offered by the Special Training Division of the Oak Ridge Institute of Nuclear Studies. Interested persons are urged to make application promptly for one of the courses in view of the heavy load of applications from university persons for participation in the summer courses. The courses will begin on January 2, January 30 and March 6, 1950. Requests for additional information should be addressed to Dr. Ralph T. Overman, Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tenn. The courses are offered at Oak Ridge to acquaint research workers with the safe and efficient use of radioisotopes as tracers. Emphasis is placed on individual laboratory work, with sufficient lecture material included to provide the necessary background information. Eleven of the courses have already been given. Thirty-two participants can be accommodated in each of the three four-week courses.

DR. CHAPMAN SUCCEEDS DR. BOWERS AS EXECUTIVE DIRECTOR

The chairman of the Committee on Human resources of the Research and Development Board, Department of Defense, announces the appointment of Dwight W. Chapman, Ph.D., as executive director of the committee to succeed Raymond V. Bowers, Ph.D., who has accepted the directorship of the Air University Human Resources Research Institute, Maxwell Field, Montgomery, Ala. Dr. Chapman has been associated with the committee since 1947; during the war he was acting director of Civilian Surveys for the War Production Board and helped develop the wartime selection program of the Office of Strategic Services.

Medical News

(Physicians will confer a favor by sending for this department items of news of general interest: such as relate to society activities new hospitals, education and public health. Programs should be received at least two weeks before the date of meeting.)

CONNECTICUT

Institute of Living.—About $100,000 has been donated by friends to the Institute of Living at Hartford for the erection of three new cottages, which will be in the Tudor style and of fire-proof construction. In each cottage there will be a combination gymnasium and recreation room and modern hydrotherapy facilities. Occupancy is scheduled for January and February, 1950.

Institute on Maternity Nursing.—The Yale University School of Nursing and the Connecticut State Department of Health will conduct an Institute on Maternity Nursing November 28-December 2. The institute will include lectures and small discussion groups, mornings and afternoons. Emphasis will be placed on some of the recently developed procedures, such as natural childbirth, rooming-in, early ambulation, self-demand feeding and parents' classes. Participants in the institute, which will be held in the Yale School of Nursing, will be: Dr. Herbert Thoms, professor of obstetrics and gynecology at Yale University School of Medicine, New Haven; Dr. Grover F. Powers, professor of pediatrics; Dr. Martha Clifford, of the Bureau of Maternal and Child Hygiene, Connecticut State Department of Health; Miss Hazel Corbin, director of the Maternity Center Association, and Miss Kate O. Hyder, associate professor of obstetric nursing at Yale. A limited number of tuition stipends for the institute will be granted to nurses employed in Connecticut. They are available through the Connecticut State Department of Health. Applications should be addressed to Dr. Martha Clifford, State Department of Health, Hartford. Enrolment in the institute will be limited to 50 persons. Those wishing to enroll should write to the Office of the Dean, Yale University School of Nursing, 310 Cedar St., New Haven.

DELAWARE

State Medical Election.—At the annual session of the Medical Society of Delaware October 10-12, the following officers were elected for the year 1950: Drs. Carl B. Scull, Dover, president; Charles E. Wagner, Wilmington, president-elect; Andrew M. Gehret, Wilmington, secretary, and Joseph M. Messick, Wilmington, treasurer. Dr. James Beebe, Lewes, was elected delegate to the American Medical Association with Dr. Wagner as alternate.

ILLINOIS

Televise Fluoroscopic Image of Heart.—On November 30 the Educational Committee of the Illinois State Medical Society will televise over WGN-TV a fluoroscopic image of a living heart. The recent development of a specially sensitized camera tube has made the demonstration possible. Participants in the November 30 telecast, titled "So You've Had a Heart Attack," will be Chicago physicians, Dr. John A. Mart, instructor in medicine at Northwestern University Medical School, and Dr. Theodore R. Van Dellen, assistant dean of the school and medical editor of the *Chicago Daily Tribune*.

Chicago

Society News.—At the recent annual meeting of the Chicago Society of Internal Medicine, the following officers were elected: Drs. Sidney Strauss, president; Howard L. Alt, vice-president, and Ernest G. McEwen, secretary-treasurer.

Dr. Harris Named Director of VD Clinics.—Dr. William W. Harris, assistant to medical director of the Intensive Treatment Center since 1946, has been named director of clinics of the Chicago venereal disease control program. He will direct the four diagnostic and treatment centers.

Children's Neurologic Service Ward.—A children's ward in the neurologic service has been opened at the University of Illinois Neuropsychiatric Institute. The new ward will be under the supervision of Dr. Eric Oldberg, professor of neurology and neurologic surgery and head of the department at the University of Illinois College of Medicine.

Grant to Epilepsy Clinic.—The Junior League of Chicago has awarded a gift of $13,000 to the Research and Educational Hospitals for the purpose of maintaining and operating the Consultation Clinic for Epilepsy. The gift covers a period of one year and represents the continuation of support of the clinic by the Junior League. More than 1,000 patients have been seen in the clinic since it was established in 1946. The Division of Rehabilitation of the State Department of Public Welfare and the Division of Special Services for Crippled Children have also contributed to the clinic's support. Dr. Frederic A. Gibbs is director.

Solomon Research Foundation.—A new $35,000 research laboratory has been completed at the Hektoen Institute of Cook County by the Dr. Jerome D. Solomon Medical Research Foundation, which was established in 1944 in memory of Captain Jerome D. Solomon of the Army Medical Corps, who died of scrub typhus while on duty in the Southwest Pacific area.

Personals.—Dr. Bowman C. Crowell of Chicago was guest of honor at a farewell dinner at the Drake Hotel November 12 given by the staffs of the American College of Surgeons and of *Surgery, Gynecology and Obstetrics*. Dr. Crowell, a pathologist, is retiring as associate director and director of clinical research of the college, with which he has been connected since 1926. Dr. and Mrs. Crowell plan to spend the winter in Florida. ——Dr. Samuel J. Taub recently addressed the staff of the Hôpital Policlinique des Enfants in Paris, France, on allergic manifestations of the eye and surrounding structures and on superficial punctate keratitis with allergic causation.

INDIANA

Expand Full Time Staff.—Further expansion of the full time staff of the Indiana University School of Medicine on the Medical Center campus at Indianapolis and the University campus at Bloomington to meet an increased student enrolment and a growing research program has been announced. Recent appointments include: Dr. Herbert S Gaskill as professor of psychiatry. Formerly on the staff of the University of Pennsylvania and a member of the psychiatric department of the university hospitals, Dr. Gaskill will integrate psychiatric teaching and consultation into the teaching of medicine, surgery, obstetrics and pediatrics. During World War II he was chief of neuropsychiatry with the 20th General Hospital and later was assigned to the Surgeon General's office. Dr. Edward W. Shrigley, former assistant professor of bacteriology at Yale University, New Haven, Conn., as associate professor of microbiology. In addition to his teaching in the medical school, Dr. Shrigley will continue his investigative work. Dr. Philip F. D. Seitz as assistant professor of psychiatry and director of psychiatric research. Dr. Seitz was formerly on the staff of the University of Pennsylvania and psychiatrist to the Skin and Cancer Hospital at Philadelphia. During World War II he was chief of neuropsychiatry with the 280th General Hospital and chief of medicine with the 319th Station Hospital.

Appointments to the department of anatomy staff of the School of Medicine at Bloomington include Lawrence O. Morgan, Ph.D., for 20 years associate professor of anatomy, histology and neurology at the University of Cincinnati, as professor.

KENTUCKY

Director of Blood Center Appointed.—Dr. Marion F. Beard has been appointed medical director of the Louisville Regional Blood Center. Dr. Beard, who is an assistant professor of medicine at the University of Louisville School of Medicine, formerly was research instructor at Ohio State University School of Medicine, Columbus.

New 125 Bed Psychiatric Wing.—The new $1,000,000 psychiatric wing of the Norton Memorial Infirmary, Louisville, is now in full operation with the opening of its fifth floor. The 125 bed, six floor addition consists of wards for alcoholic, psychiatric and psychosomatic cases, with conference rooms and recreation quarters on the top floor. This division of the hospital is a teaching facility of the University of Louisville School of Medicine.

LOUISIANA

University Appointment.—Dr. Ian P. Stevenson, who recently completed training as a fellow in psychosomatic medicine at Cornell University, New York, has been appointed assistant professor of neuropsychiatry of medicine at Louisiana State University School of Medicine, New Orleans.

Reestablish Social Service Department.—The department of social service has been reestablished in the Tulane University of Louisiana School of Medicine, New Orleans, to help patients with environmental and personal problems related to their illness. Patients entering Tulane clinics will be examined routinely by clinicians, who, if they discover personal, social or environmental problems, will refer the patients to the social service department.

MICHIGAN

Dr. Vaughan Receives Sedgwick Medal. — Henry Vaughan, D.Ph., dean of the School of Public Health at the University of Michigan, was honored at a banquet of the American Public Health Association at the Hotel Statler in New York the week of October 23. Dr. Vaughan was Detroit Commissioner of Health for 23 years before he left in 1941 to head the public health school at Michigan. The Sedgwick award was established in 1929 to honor the memory of William Thompson Sedgwick, for 38 years professor of biology and public health at the Massachusetts Institute of Technology.

Cancer Diagnostic Clinic.—Establishment of a cancer diagnostic-teaching clinic for the instruction of medical graduates has been made a cooperative program of the American Cancer Society, the Detroit Institute of Cancer Research and the Wayne University College of Medicine, Detroit. Facilities for teaching special technics in cancer detection to 50 physicians a year will be provided. The college will undertake staffing and operating the clinic, principal facilities of which will be located in the Cancer Center Building, John R. and Hancock streets. Facilities of the Tumor Clinic of City of Detroit Receiving Hospital and the Tumor Board of the Veterans Administration Hospital in Dearborn will also be used. The program can be managed with funds now available to the college through grants-in-aid for cancer teaching and other funds to be supplied by the American Cancer Society. Personnel help will also be underwritten by the Veterans Administration Hospital.

MINNESOTA

Course in Obstetrics.—The University of Minnesota announces a continuation course in obstetrics, December 16-17, for general practitioners. Etiology, diagnosis and management of obstetric complications will be presented by means of lectures and round tables.

Society News.—The roster of officers elected at the meeting of the Southern Minnesota Medical Association on Monday, September 12, at Red Wing, includes Dr. Warren E. Wilson, Northfield, president, and Dr. Wallace A. Merritt, Rochester, secretary-treasurer.

NEW YORK

Survey by Water Pollution Board. — The Roundout-Wallkill drainage basin in Ulster County is the site of the first stream survey to be conducted for the recently established New York State Water Pollution Control Board. This survey is intended to demonstrate the operating procedures and technics proposed for all such pollution investigations and to provide experience for survey personnel. In addition, the field data now being obtained will be used in determining the actual classification of the waters in this basin after public hearings at a later date. This watershed was chosen because it is a representative drainage area of 210 square miles. The U. S. Public Health Service has made a mobile laboratory available. The Water Pollution Control Act, passed at the last session of the legislature, provides for the classification of New York waters and the establishment of standards of quality and purity according to the best uses in the interests of the public to which the water is adapted. This survey is being carried out under the direction of Mr. Charles C. Agar, chief of the Water Pollution Control Section of the department, and under the general supervision of A. F. Dappert, executive secretary of the New York State Water Pollution Control Board.

New York City

Personals.—Dr. Emanuel D. Friedman, professor emeritus of neurology, New York University-Bellevue Medical Center, College of Medicine, received the Townsend Harris Medal September 22 at the sixty-ninth annual dinner of the City College Alumni.——Lester R. Cahn, D.D.S., will read a paper entitled "Virus Infections of the Mouth" at the monthly conference of the New York Institute of Clinical Oral Pathology, November 28 at the New York Academy of Medicine.

Lowenstein Memorial Pavilion. — Dedication ceremonies were held October 23 at the Hillside Hospital, Bellerose, Long Island, N. Y., for the dedication of the new Lowenstein Memorial Pavilion, which increases the hospital bed capacity from 88 to 175. Hillside Hospital is a nonprofit and nonsectarian mental institution for the treatment of patients suffering from early and curable mental symptoms, regardless of their ability to pay. It is also a training institute for physicians in postgraduate psychiatry and psychotherapy. At present there are 12 full time residents in training. Since 1948 it has been an affiliate of the Federation of Jewish Philanthropies of New York.

OHIO

Cleveland's Health Center.—The Newton D. Baker Health Center was dedicated October 18 as Cleveland's first real "health center." It has been under construction since Sept. 16, 1947. It will serve as headquarters for the Division of Health Personnel and a meeting place for health education groups. Tuberculosis diagnostic service and child welfare clinics have been established, and other clinics will be set up as need is shown.

Personals. — Dr. Herbert B. Wright, Cleveland, recently became president of the Cleveland Academy of Medicine.—— Dr. Robert S. Dinsmore has been appointed to succeed the late Dr. Thomas E. Jones as chief of staff of surgery at Cleveland Clinic. Dr. Dinsmore has been a member of the clinic's surgical staff since 1924. He received his medical degree at Washington University School of Medicine, St. Louis, 1917. He is a member of the American Surgical Association, Eastern Surgical Society, the American Association for the Study of Goiter and many other organizations.

OREGON

State Medical Election.—The Oregon State Medical Society at its meeting in Eugene October 12-14 elected the following officers: Drs. James E. Buckley, Portland, president; William J. Weese, Ontario, president-elect; Werner E. Zeller, Portland, secretary, and Robert W. Kullberg, Portland, treasurer.

UTAH

University Appointments.—Dr. Hyrum L. Marshall, professor and head of the department of public health and preventive medicine, has been named acting dean of the University of Utah College of Medicine, Salt Lake City. Dr. James F. Bosma, formerly assistant professor of pediatrics at the University of Minnesota Medical School, Minneapolis, has been appointed professor and head of the department of pediatrics, succeeding Dr. John A. Anderson. Dr. Leonard H. Taboroff, Topeka, Kan., has assumed the duties of director of the new Utah Child Guidance Center, which is to be administered under the departments of psychiatry and pediatrics of the university. Dr. Taboroff, who will be assistant professor in psychiatry and pediatrics, received his training in child psychiatry in the Children's Division (Southard School) of the Menninger Foundation.

VERMONT

University Lectures on Growth and Malignancy.—A series of meetings on problems of growth and malignancy will be presented throughout the winter by the department of pathology of the College of Medicine at the University of Vermont, Burlington. Meetings will be at 8 p. m. in the Fleming Museum. Dr. Charles B. Huggins, professor of surgery, University of Chicago, will speak December 2 on "Serum Proteins in Cancer" at a joint meeting with the Osler Society. On Jan. 5, 1950, Leonell C. Strong, Ph.D., professor of anatomy at Yale University School of Medicine, New Haven, Conn., will speak on "Genetics and Cancer," and on February 9, Dr. Albert Tannenbaum, Chicago, will speak on "Diet and Cancer." The speaker March 2 will be Van R. Potter, Ph.D., professor of oncology, University of Wisconsin Medical School, Madison, whose subject will be "Enzymes, Growth and Cancer." At a joint meeting with the Chittenden County Medical Society March 30, 1950, Dr. Roy Hertz, chief of the Endocrinology Section of the National Cancer Institute, Bethesda, Md., will talk on "Vitamin-Hormone Interrelationships Affecting Tissue Growth." The final speaker in the series on May 4, will be Dr. Robert E. Stowell, professor of oncology, University of Kansas School of Medicine, Kansas City, whose subject will be "Nucleoproteins, Growth and Cancer."

WEST VIRGINIA

Mental Hygiene Service.—A diagnostic service to detect emotional and behavior problems has been inaugurated in Fayette County by the Bureau of Mental Hygiene of the State Department of Health. A psychiatrist, psychologist and social worker will visit the Fayette Clinic two days each month to review cases screened by the professional personnel of the local unit. Certain cases will be referred to one of the four existing mental clinics in the state. The Wood county clinic, Parkersburg, is another recent addition to the mental hygiene program in West Virginia. This free child guidance clinic will be sponsored locally for the first two years. Through the effort of the state department of health, mental hygiene facilities are now being offered in five areas with clinics in Charleston, Kanawha County; Martinsburg, Berkeley County; Huntington, Cabell County, and Parkersburg, Wood County.

WISCONSIN

State Medical Election.—At the annual meeting of the State Medical Society of Wisconsin in Milwaukee October 3-5 the following officers were elected: Drs. John W. Truitt, Milwaukee, president, and Henry H. Christofferson, Colby, president-elect; Dr. William D. Stovall, Madison, was reelected delegate to the American Medical Association and Dr. David J. Twohig Sr., Fond du Lac, was renamed alternate delegate.

Heart Research Institute.—With a grant of $291,000 from the federal government, the University of Wisconsin, Madison, will soon begin construction of a Heart Research Institute, which will consolidate all phases of cardiovascular research. Plans call for a fifth and sixth floor addition to McArdle Memorial laboratory to house this research project. The move will facilitate coordination work in heart research in the fields of physiology, pharmacology, anesthesiology, medicine, surgery and anatomy.

PUERTO RICO

Hospital Construction Assured.—An annual appropriation of $4,600,000 for hospital construction in Puerto Rico is included in Senate Bill 614, to amend the 1947 Hospital Survey and Construction Act, recently signed by President Truman. Projects planned in Puerto Rico, for which federal funds are expected, include a new mental hospital at Rio Piedras, at an estimated cost of $5,000,000, a new general district hospital at Ponce, at a cost of $3,000,000, and a new tuberculosis hospital. Construction of a new tuberculosis hospital was started at the beginning of this year at Rio Piedras, Puerto Rico. The Insular Legislature has already approved legislation to match federal funds, on the basis of the previous law.

GENERAL

Society Meeting.—The Association for Research in Nervous and Mental Disease will hold its annual meeting at the Hotel New Yorker, New York, December 2-3 on "Life Stress and Bodily Disease" under the presidency of Dr. Harold G. Wolf, New York.

Prize for Best Article.—A prize is being offered by the Gynecology Travel Club of Phi Delta Epsilon for the best article on the subject of gynecology or endocrinology related in this field. The contest is open to interns, residents and undergraduates only. The first award will be made at the 1950 convention, and articles will be accepted until Oct. 1, 1950. Submit entries or write for further information to: Dr. Emil D. Rothman, 722 Maccabees Building, Detroit 2.

Arthritis Foundation Opens National Campaign.—General Lucius D. Clay, board chairman Floyd B. Odlum and Dr. Philip S. Hench, Rochester, Minn. of the Medical and Scientific Committee, addressed guests at a dinner at the Somerset Hotel in Boston on November 21 at 6:45 p. m., marking the opening of the 1949 national campaign of the Arthritis and Rheumatism Foundation.

Seek Funds for Crippled Children in Greece.—The Near East Foundation is seeking funds to carry on its work of rehabilitation among the crippled children of Greece. Children are suffering from malnutrition and disease and from injuries resulting from land mines planted by the occupation forces and more recently by the guerrillas. The foundation is located at 54 East 64th Street, New York 21.

Plastic Surgery Award.—The American Society of Plastic and Reconstructive Surgery offers an annual award of $250 to a young surgeon who is a citizen of the United States and who gives promise of distinguished research work in plastic and reconstructive surgery. The award will be made on the basis of a written outline of the research project to be undertaken. A report on completion of the project will be presented by the author at an annual meeting of the society. For details write Dr. Clarence R. Straatsma, Secretary, 66 East 79th Street, New York.

Electroencephalographic Society Meeting.—The Southern Electroencephalographic Society meeting will be held in New Orleans November 30. Dr. Martin L. Towler will give the presidential address and will also present a paper on the effect of phenurone on the electrocardial activity of normal subjects. Other papers to be presented include electroencephalography in infants during sedation sleep, value of sleep studies, electroencephalograms in cases of brain tumor and electroencephalographic changes during the menstruation cycle compared with other physiologic variables. Two roundtable discussions will be presented, one concerning the general technic of electroencephalography as carried on in various laboratories in the South and a second on problems of localization of cortical damage.

Awards in Military Medicine.—Lieut. Elliott S. Hurwitt (MC, USNR), New York, was awarded the Sir Henry Wellcome Medal and a prize of $500 by the Association of Military Surgeons of the United States at their annual banquet November 11 in Washington, D. C. His paper, "A Blood Vessel Bank under Military Conditions," was selected by the association as the best article submitted in the annual competition for 1949. The award was established by the late Sir Henry Wellcome. The presentation of honorarium and scroll accompanying the award was made by Rear Admiral Joel T. Boone (MC, USN), president of the association. Dr. Hurwitt entered the Navy in 1943 and was released to inactive duty in 1946. He received his medical degree from Harvard Medical School, Boston, in 1937. Dr. H. Trendley Dean, dental director, U. S. Public Health Service, received the Gorgas Award for 1949 "for outstanding contributions in the field of military medicine." This award is sponsored by Wyeth, Inc., pharmaceutical manufacturers of Philadelphia. Dr. Dean for many years has specialized on the epidemiology of dental diseases.

National Epilepsy League Formed.—Two organizations, the American Epilepsy League and the National Association to Control Epilepsy, united last month to form the National Epilepsy League, with headquarters at 130 North Wells Street, Chicago. The new organization states as its purposes: to encourage research in the causes and prevention of epilepsy; to assist epileptic persons by compiling information on available medical resources, school facilities and job opportunities, and to dissolve the stigma associated with epilepsy by focusing attention on the opportunities for control of the seizures which mark the disorder. Allied with the new organization in its program will be the nationwide service facilities of the National Society for Crippled Children and Adults. Ben H. Gray, national director of the American Epilepsy League, will continue as executive officer for the new organization. The merger was completed by Mr. and Mrs. M. Peter Miller, recently of Washington, D. C., representing the American Epilepsy League, and Mr. and Mrs. Fred X. Markham of Los Angeles, directors of the National Association to Control Epilepsy. The board of the new league will include present board members of both organizations.

Mental Health Assembly.—The Second Mental Health Assembly of World Federation for Mental Health met in Geneva, Switzerland, August 22-27, at which time it completed plans for its activities during the coming year. The United States membership includes the American Association of Mental Deficiency, American Association of Psychiatric Social Workers, American Neurological Association, American Nurses' Association, American Orthopsychiatric Association, Inc., American Psychiatric Association, American Psychoanalytic Association, National Committee for Mental Hygiene, National League of Nursing Education, National Mental Health Foundation, National Organization for Public Health Nursing, Society for Applied Anthropology and Society for the Psychological Study of Social Issues. Dr. John R. Rees (England) was appointed director-general and Dr. Kenneth Soddy (England) secretary. The offices of the federation will continue to be located temporarily at 19 Manchester Street, London W. 1, England. The new president of the federation is Dr. Andre Repons of Switzerland, who replaces Dr. Rees. Dr. Frank Fremont-Smith (U. S. A.) has submitted his resignation as treasurer and Dr. M. K. el Kholy of Egypt has accepted the post of acting treasurer. Dr. George S. Stevenson, New York, medical director of the National Committee for Mental Hygiene, remains as a member of the executive board, and Dr. Leo H. Bartemeier of Detroit has been appointed to the executive board as alternate.

Institute of Nutrition Awards.—The American Institute of Nutrition invites nominations for the following awards to be made at its annual meeting next spring: The Osborne and Mendel Award of $1,000 established by the Nutrition Foundation Inc. for the recognition of outstanding accomplishments in the general field of exploratory research in the science of nutrition. It will be given to the investigator who, in the opinion of the jury of award, has made the most significant published contribution in 1949 or who has published a series of contemporary papers of outstanding significance. Normally preference will be given to research workers in the United States and Canada, but investigators in other countries, especially those sojourning in the United States or Canada for a period of time, are not excluded from consideration.

The 1950 Award of $1,000 established by Mead Johnson & Company to promote researches dealing with the B complex vitamins. The recipient of this award will be chosen by a Committee of Judges of the American Institute of Nutrition. The award will be given to the laboratory or clinical research worker in the United States or Canada who has published

during 1949 the most meritorious scientific report dealing with the field of the B complex vitamins. While the award will be given primarily for publication of specific papers, the judges are given considerable latitude in the exercise of their function. It may be recommended that the award be made to a worker for valuable contributions over an extended period but not necessarily representative of a given year.

The 1950 award of $1,000 and a gold medal made available by the Borden Company Foundation, Inc. The American Institute of Nutrition will make this award in recognition of distinctive research by investigators in the United States and Canada which has emphasized the nutritive significance of the components of milk or of dairy products. The award will be made primarily for the publication of specific papers, but the judges may recommend that it be given for important contributions over an extended period of time. The award may be divided between two or more investigators. Employees of the Borden Company are not eligible for this honor.

The nominations should be accompanied with such data relative to the nominee and his research as will facilitate consideration for the award. Nominations for any of the three awards must be in the hands of the chairman of the nominating committees by Jan. 15, 1950: Osborne and Mendel Award, H. E. Carter, University of Illinois, Urbana; "B-Complex" Award, W. H. Sobrell Jr., Experimental Biology and Medicine Institute, National Institutes of Health, Bethesda, Md., and the Borden Award, L. A. Maynard, Cornell University, Ithaca, N. Y.

Prevalence of Poliomyelitis.—Reports of cases of poliomyelitis for the periods indicated have been received from the National Office of Vital Statistics, U. S. Public Health Service.

Division and State	Week Ended Nov. 12, 1949	Week Ended Nov. 13, 1948	Median 1944-1948	Jan. 1 to Nov. 12, 1949	Jan. 1 to Nov. 13, 1948
North Eastern States					
Maine	6	..	..	449	38
New Hampshire	1	..	1	182	18
Vermont	2	..	1	140	22
Massachusetts	25	1	12	1,753	182
Rhode Island	1	..	..	162	8
Connecticut	14	1	5	694	114
Middle Atlantic States					
New York	94	20	31	5,292	1,384
New Jersey	35	15	9	1,438	772
Pennsylvania	26	13	14	789	706
East North Central States					
Ohio	21	19	19	1,704	1,121
Indiana	24	5	3	1,088	380
Illinois	46	19	19	2,795	1,055
Michigan	63	21	16	2,692*	712
Wisconsin	8	20	20	1,018	589
West North Central States					
Minnesota	23	30	16	1,826	1,262
Iowa	24	28	12	1,092	1,112
Missouri	11	8	8	1,300	300
North Dakota	3	2	..	443	117
South Dakota	23	60	..	376	727
Nebraska	18	11	6	635	685
Kansas	9	10	2	703	303
South Atlantic States					
Delaware	..	..	..	43	129
Maryland	16	5	4	265	112†
District of Columbia	2	2	1	104	128
Virginia	8	12	3	325	551
West Virginia	5	3	3	340	162
North Carolina	1	20	4	216*	2,439†
South Carolina	1	4	..	101	362
Georgia	8	..	2	219	226
Florida	5	8	4	242	258
East South Central States					
Kentucky	29	2	2	636	195
Tennessee	14	5	5	522	364
Alabama	3	2	2	236	202
Mississippi	17	1	2	333	149
West South Central States					
Arkansas	8	3	2	959	137
Louisiana	4	..	3	220	138
Oklahoma	15	5	3	1,255	349
Texas	29	27	9	2,204	1,663
Mountain States					
Montana	2	1	1	93	69
Idaho	4	2	2	403	106
Wyoming	4	2	..	107*	78
Colorado	6	2	2	639	118†
New Mexico	1	2	1	195	77
Arizona	5	7	1	154	152
Utah	8	26	2	268	139
Nevada	1	1	..	16	8
Pacific States					
Washington	11	7	7	566	327
Oregon	10	9	5	285	171
California	66	198	34	2,300	2,624
Total	751	669	...	39,792*	25,140†
Median, 1944-1948	...	...	330	18,202	

* Deductions: Michigan, weeks ended August 20 and October 22, 1 case each; North Carolina, week ended October 15, 1 case, and Wyoming, week ended October 1, 1 case.
† Figures changed by corrected reports.

CANADA

Investigation of Chiropractors.—A commission was appointed by the Government of the Province of Quebec to carry out a judicial investigation on chiropractics. After a study of several months, the commission concluded: (*a*) That the chiropractors have failed entirely in their task to establish that their concepts have a scientific base; (*b*) that their practice of manipulations on the vertebral column is useless in most of the cases and dangerous in a large number of them; (*c*) that the teaching dispensed in the existing school of chiropractics is, according to testimonies, notoriously inferior to reasonable standards and that this was admitted by the chiropractors themselves, and (*d*) that the province possesses institutions highly qualified for medical teaching and there does not appear any valid reason to permit the art of healing to be taught in schools infinitely less qualified and located outside the province and consequently outside the control of the provincial government.

LATIN AMERICA

Society News.—The Venezuelan Pediatric Society has chosen the following officers for 1949-1951: president, Dr. Lya Imber de Coronil; secretary, Dr. Guillermo Tovar, P. O. Box 5, Caracas, and treasurer Dr. Miguel Ragá.

Surgical Societies Merge.—The Sociedad de Cirujanos de Chili (The Society of Surgeons of Chile) has recently been established from fusion of the Society of Hospital Surgeons and the Society of Surgery of Chile. Officers for the year 1949-1950 are Dr. Gustavo Maturana B., president, and Dr. Leonidas Aguirre Mac-Kay, Santiago, secretary.

Society of Anesthesiology.—The first Cuban Society of Anesthesiology was organized in September at the suggestion of Dr. Angel Pérez Andre, director of the postgraduate courses in medicine and surgery at the University of Havana. The officers of the society are: Drs. J. L. Avellanal, president; Ramón Alvárez, secretary, and Juan F. Sánchez Casanova, treasurer.

Congresses.—The twelfth International Congress of Medicine and Pharmacy was held in Mexico City on October 23 to 29. The main topic was "Safety of the Future of Medicine During War." Dr. J. Voncken, who was invited by the Faculty of Laws of the University of Mexico, spoke on "Medical International Rights." After the congress an important session on the aforementioned subject was held in Havana. Dr. Voncken was invited to give lectures in the United States.

DEATHS IN FOREIGN COUNTRIES

Prof. Vincento Rossi, chairman of the medical faculty of Pisa University, died in Pisa June 28, aged 59.——**Dr. Alfred Ernest Barclay,** dean of British radiology and president emeritus of the Sixth International Congress of Radiology to be held in London, 1950.——**Dr. Otto Kauders,** Vienna, chairman and professor of the psychiatric-neurologic clinic at the University of Vienna died August 6.——**Dr. Schack August S. Krogh,** Danish scientist, physiologist, winner of the Nobel Prize in 1920 for experiments on the function and regulation of the capillaries and professor at the University of Copenhagen, died September 13, aged 74.——**Dr. William G. Willoughby,** past president of the British Medical Association and medical officer of health for Eastbourne for 45 years, died August 13, aged 83.

Marriages

JOHN ROBERT KERSTEN, Fort Dodge, Iowa, to Miss Martha Jeanne Montgomery on Governor's Island, N. Y., September 17.

HARRY FREDERICK KAACK JR., Clinton, Iowa, to Miss Elizabeth Louise Johnson of Davenport, September 24.

PHILIP B. CHASE, Farmington, Maine, to Miss Kathleen L. Knippel of West Bend, Wis., September 22.

WARREN LEROY HOLLIS, Lacona, N. Y., to Miss Betty Jane Eicholzer, of Syracuse, September 20.

PHILIP DAVID COVNER, Lynn, Mass., to Miss Shirley Annette Chaves of Swampscott, September 18.

WILLIAM C. PRITCHARD to Miss Betty L. Hay, both of Portland, Ore., October 1.

HYMAN MERLIN to Miss Ester Argintar, both of Miami, Fla., September 18.

Deaths

Charles Anderson Aldrich ⊕ Rochester, Minn.; born in Plymouth, Mass., March 4, 1888; Northwestern University Medical School, Chicago, 1915; professor of pediatrics at the University of Minnesota Graduate School and director of the Rochester Child Health Project; associate in pediatrics, 1934-1935, assistant professor, 1935-1936 and professor of pediatrics from 1941 to 1944 at Northwestern University Medical School in Chicago, where he was associated with the Children's Memorial Hospital for many years, serving as chief of staff from 1941 to 1944; for many years associated with the Evanston (Ill.) Hospital; secretary of the Section on Diseases of Children of the American Medical Association from 1927 to 1930 and chairman 1930-1931; on advisory Board of Medical Specialties, 1935-1947; president in 1946 and member of the council from 1935 to 1942 of the American Pediatric Society; past president of the Chicago Pediatric Society and the Evanston Branch of the Chicago Medical Society; member of the American Academy of Pediatrics, American Psychosomatic Society, Society for Research in Child Development, National Association for Nursery Education, Minnesota Mental Hygiene Society and White House Conference on Children in a Democracy; honorary member of the National Committee on Mental Hygiene, Northwest Pediatric Society and Omaha Mid-West Clinical Society; in 1933 secretary of the American Committee of the third International Pediatric Congress in Stockholm, Sweden; secretary-treasurer from 1934 to 1944 and president from 1945 to 1947 of the American Board of Pediatrics; from 1941 to 1944 member of the committee, Elizabeth McCormick Child Research Grant and trustee of the Illinois Children's Home Aid Society; served as a member of the educational council of the Winnetka Public Schools and as member of the school board; member of the board of North Shore Country Day School; received the Lasker Award in Mental Health for 1948, the citation reading in part "for outstanding accomplishment in the education of physicians in the psychological aspects of the practice of medicine through a community wide child health program in Rochester, Minn."; author of "Cultivating the Child's Appetite" (1927); joint author with Mary M. Aldrich of "Babies are Human Beings" (1938) and "Feeding Our Old Fashioned Children" (1941); contributed numerous professional articles and books reviews to medical periodicals; on the editorial board of *Pediatrics;* died in St. Mary's Hospital October 5, aged 61, of carcinoma.

Murdoch Bannister ⊕ Ottumwa, Iowa; born in Detroit Dec. 25, 1858; University of Pennsylvania Department of Medicine, Philadelphia, 1894; past president of the Wapello County Medical Society; member of the Des Moines Valley Medical Society; fellow of the American College of Surgeons; served during World War I; member of the board of control of Iowa state institutions from 1910-1913; civil areonautics administration examiner at Ottumwa for many years and a surgeon for the Chicago, Burlington and Quincy Railroad; member of the board of directors of the Fidelity Savings Bank; affiliated with St. Joseph's Hospital and the Ottumwa Hospital, where he died September 21, aged 80.

Frederic Augustus Alling ⊕ Newark, N. J.; born in Newark, N. J., in 1884; Columbia University College of Physicians and Surgeons, New York, 1911; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; served in France during World War I; affiliated with the Essex County Sanatorium, Verona, Rahway (N. J.) Memorial Hospital, Mountainside Hospital, Montclair, and the Orange (N. J.) Memorial Hospital, Hospital of St. Barnabas and for Women and Children, Newark Eye and Ear Infirmary, Presbyterian Hospital and the Newark City Hospital; died at Montclair October 20, aged 65.

Harvey Gamaliel Parker, Santa Ana, Calif.; born in Owatonna, Minn., Sept. 5, 1877; University of Minnesota College of Medicine and Surgery, Minneapolis, 1901; for many years practiced in Portland, Ore., where he was professor and head of the department of dermatology in the University of Oregon Medical School and associated with Multnomah Hospital; member of the Oregon State Medical Society and the Portland Academy of Medicine; member of the founders group of the American Board of Dermatology and Syphilology; died in St. Joseph's Hospital October 8, aged 72, of cardiovascular disease.

⊕ Indicates Fellow of the American Medical Association.

Charles Christian Anderson, Little Rock, Ark.; University of Arkansas School of Medicine, Little Rock, 1900; member of the American Medical Association; died August 16, aged 72, of coronary artery occlusion.

Edwin Lewis Apperson, Denver; Washington University School of Medicine, St. Louis, 1902; served on the staffs of the Presbyterian, National Jewish, Childrens and Colorado General hospitals; died September 29, aged 71, of cor pulmonale.

Herman Louis Balamuth, New York; University and Bellevue Hospital Medical College, New York, 1909; died in September, aged 61.

Joseph Randolph Barr, Tulelake, Calif.; Willamette University Medical Department, Salem, Ore., 1907; served during World War I; died August 27, aged 67.

David Major Bell, Canonsburg, Pa.; Jefferson Medical College of Philadelphia, 1903; member of the American Medical Association; served on the school board; on the staff of Canonsburg Hospital; died September 8, aged 73, of cerebral hemorrhage.

Henry Sumner Bennett ⊕ Moline, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1901; formerly member of the board of education; served during World War I; died in the Moline Public Hospital September 25, aged 73, of prostatic hypertrophy and nephrosis.

Bradford Bissell, Fort Thomas, Ky.; Duke University School of Medicine, Durham, N. C., 1936; member of the Medical Society of the State of New York and the American Medical Association; served during World War II; certified by the National Board of Medical Examiners; died near Covington September 24, aged 41, in an automobile accident.

Luke Martial Boudreaux, Erath, La.; Tulane University of Louisiana School of Medicine, New Orleans, 1913; died September 28, aged 63, of acute coronary thrombosis.

Clarence Anglim Bradley, Beatrice, Neb.; College of Physicians and Surgeons, Keokuk, Iowa, 1880; member of the American Medical Association; past president of the Gage County Medical Society; for many years chairman of the Gage County Insanity Board; served on the staff of the Lutheran Hospital; died in Norfolk September 21, aged 90, of senility.

Henry Augusta Britt, Snyder, Okla.; College of Physicians and Surgeons, Little Rock, Ark., 1911; died in Frederick September 18, aged 70, of a cerebrovascular accident.

W. H. Brown, Lovelady, Texas; Central Medical College of St. Joseph, Mo., 1905; died September 23, aged 82, of uremia.

Enoch Robinson Bush, Winchester, Ky.; Kentucky University Medical Department, Louisville, 1906; member of the American Medical Association; served during World War I; died in Clark County Hospital September 20, aged 68, of carcinoma of the stomach.

Frank Allison Clawson, Meadville, Pa.; Chicago Homeopathic Medical College, 1899; the Hahnemann Medical College and Hospital, Chicago, 1905; member of the American Medical Association; associated with Spencer Hospital and City Hospital, where he died September 28, aged 71, of acute appendicitis.

Linne H. Corson ⊕ Cumberland, Md.; College of Physicians and Surgeons, Baltimore, 1915; affiliated with the Allegany Hospital of the Sisters of Charity; died in Memorial Hospital September 23, aged 64, of cerebral hemorrhage.

Frederick David Culbertson ⊕ Rushville, Ill.; Northwestern University Medical School, Chicago, 1906; founder and owner of Culbertson Hospital, where he died September 7, aged 67.

Donald Austyn Curtis, Hackensack, N. J.; Long Island College Hospital, Brooklyn, 1917; member of the American Medical Association; served during World War II; assistant medical examiner for Bergen County; affiliated with Hackensack Hospital, where he died September 7, aged 53, of myocardial infarction following an operation.

Simon Davis, Brooklyn; Long Island College Hospital, Brooklyn, 1904; member of the American Medical Association; on the staffs of Williamsburgh and St. Catherine's hospitals; died September 26, aged 67, of coronary thrombosis.

Alfred Fred Eckert, Hecker, Ill.; St. Louis University School of Medicine, 1910; died September 20, aged 69, of carcinoma.

George M. Evans, Philadelphia; Meharry Medical College, Nashville, Tenn., 1906; died in Pleasantville, N. J., September 5, aged 68, of cerebral hemorrhage and hypertensive heart disease.

Arthur M. Faught, Los Angeles; Lincoln (Neb.) Medical College of Cotner University, 1905; member of the American Medical Association; died September 26, aged 66.

Alexander Bailey Field, Forest River, N. D.; Victoria University Medical Department, Coburg, Ontario, Canada, 1891; member of the American Medical Association; died August 29, aged 85.

William Edward Fitch, Burlington, N. C.; College of Physicians and Surgeons, Baltimore, 1891; died in Coral Gables, Fla., September 12, aged 82.

William Wilmer Frank, Portland, Ore.; Omaha Medical College, 1898; served during World War I; for many years affiliated with the Veterans Administration Hospital, where he died September 26, aged 73, of carcinoma of the of the prostate with metastases.

Grover Cleveland Franklin, Miami, Fla.; University of Alabama School of Medicine, 1907; served during World War I; affiliated with Jackson Memorial Hospital; died September 18, aged 64, of coronary thrombosis and hepatic cirrhosis.

James E. Gill ✠ Medical Director, Captain, U. S. Navy, retired, Peoria, Ill.; Georgetown University School of Medicine, Washington, D. C., 1901; fellow of the American College of Surgeons; entered the U. S. Navy in 1903 and retired Sept. 1, 1942; died in the U. S. Naval Hospital, Bethesda, Md., September 28, aged 71, of carcinoma of the larynx and bronchopneumonia.

Orval E. Glick ✠ Kentland, Ind.; the Hahnemann Medical College and Hospital, Chicago, 1901; on the staff of St. Elizabeth Hospital, Lafayette; died August 25, aged 75, of cerebral hemorrhage.

John Gromann ✠ Utica, N. Y.; Jefferson Medical College of Philadelphia, 1900; fellow of the American College of Surgeons; affiliated with St. Luke's Hospital, where he died September 23, aged 72, of cerebral thrombosis and arteriosclerosis.

Alfred Gilbert Grunwell ✠ Lieutenant Commander, U. S. Navy, retired, Punta Gorda, Fla.; Columbian University Medical Department, Washington, D. C., 1895; entered the U. S. Navy in July 1898; retired Feb. 18, 1910; died in the U. S. Naval Hospital, Beaufort, S. C., September 18, aged 76.

John Havelock Gunter ✠ Philadelphia; Temple University School of Medicine, Philadelphia, 1917; also a dentist; professor of oral surgery at the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania; served with Canadian Army Dental Corps during World War I; associated with the Philadelphia General, Jewish, Episcopal, Oncologic and Jeanes hospitals; associate editor of *Philadelphia Medicine;* died in the Urologic Clinic-Hospital September 23, aged 61, of heart disease.

Dan Eugene Hardie, Miami, Fla.; St. Louis University School of Medicine, 1930; served during World War II; died September 22, aged 44.

Francis Bernard Hart, Brooklyn; Columbia University College of Physicians and Surgeons, New York, 1905; specialist certified by the American Board of Pediatrics; served during World War I; affiliated with the Hospital of the Holy Family, Shore Road Hospital and St. Mary's Hospital; consultant for Dykes Heights Home for Blind Children; died September 22, aged 68, of coronary thrombosis.

Samuel Ringgold Harwood, Sullivan, Mo. (licensed in Missouri in 1898); member of the American Medical Association; died September 22, aged 82.

James Tuley Hazel, Freedom, Ind.; Hospital College of Medicine, Louisville, 1900; member of the American Medical Association; served during World War I; died September 18, aged 80, of diabetes mellitus.

William Renick Henderson, Houston, Texas; Kansas City (Mo.) Medical College, 1897; died September 2, aged 77.

Frank M. Holloman, Morgan City, Miss.; Memphis (Tenn.) Hospital Medical College, 1903; member of the American Medical Association; died September 29, aged 71.

John Walker H. Holt, Asheville, N. C.; Meharry Medical College, Nashville, Tenn., 1917; affiliated with the Asheville Colored Hospital; died September 23, aged 61, of carcinoma of the lung.

E. T. Hudnall, Taylor, Ark. (licensed in Arkansas in 1903); died September 25, aged 70.

Stanley Wilton Johns, Good Water, Ala.; Atlanta School of Medicine, 1913; died August 21, aged 60, of coronary occlusion.

Thomas Ralph Kemmer, Grand Rapids, Mich.; Vanderbilt University School of Medicine, Nashville, Tenn., 1918; member of the American Medical Association; served during World War I; on the staff of Butterworth Hospital, where he died September 8, aged 57, of arteriosclerotic heart disease.

Guy A. Longbrake, Fort Myers, Fla.; Rush Medical College, Chicago, 1897; died July 28, aged 77, of cerebral hemorrhage and arteriosclerosis.

Elias Homer Loofbourrow ✠ Northfield, Minn.; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1906; died September 16, aged 71, of cardiac failure.

William Clinton Marett, Seneca, S. C.; University of Maryland School of Medicine, Baltimore, 1911; served during World War I; died September 28, aged 63.

Charles LaFayette Perry, Philadelphia; (licensed in Mississippi in 1925); died August 26, aged 72, of arteriosclerosis and myocardial failure.

Frank Wesley Phelps ✠ Seattle; Northwestern University Medical School, Chicago, 1904; an Associate Fellow of the American Medical Association; fellow of the American College of Surgeons; associated with Virginia Mason Hospital; died September 5, aged 71, of coronary thrombosis.

Joseph Adelard Rene, La Canada, Calif. (licensed in Wisconsin in 1899); served during World War I; died in Wadsworth Hospital, Sawtelle, August 31, aged 80, of pneumonia.

Joseph Rosenthal ✠ Brooklyn; Columbia University College of Physicians and Surgeons, New York, 1906; specialist certified by the American Board of Internal Medicine; affiliated with St. Joseph Hospital in Far Rockaway, Jewish Sanitarium for Chronic Diseases, and Brooklyn Hebrew Home and Hospital for Aged; attending physician and president of the medical board, Jewish Hospital and Maimonides Hospital, Israel Zion Division; died September 24, aged 65, of coronary thrombosis.

Norman Reuben Schneidman ✠ Minneapolis; University of Minnesota Medical School, Minneapolis, 1937; served during World War II; died in the Veterans Administration Hospital September 6, aged 42, of coronary thrombosis.

Harry A. Shute, Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1894; member of the American Medical Association; served on the staff of St. Agnes Hospital; died in Lankenau Hospital September 26, aged 86, of spontaneous left pneumothorax and bronchopneumonia.

Lawrence Henry Smith ✠ East Aurora, N. Y.; University of Buffalo School of Medicine, 1907; served during World War I; member of the staff of Buffalo General Hospital; died September 25, aged 70, of cerebral hemorrhage.

Simon Steelsmith, Abilene, Kan.; Medical College of Indiana, Indianapolis, 1881; member of the American Medical Association; died August 22, aged 93, of myocarditis.

Stephen W. Stuteville, Grand View, Ind.; University of Louisville (Ky.) Medical Department, 1893; county health officer for many years; formerly clerk of the Circuit Court; died September 30, aged 78.

Raleigh Robert Sullivan ✠ Lakeland, Fla.; University of Louisville (Ky.) Medical Department, 1892; associated with Morrell Hospital; died September 11, aged 79, of acute myocardial infarction.

Joseph Rush Tanner, Chicago; Meharry Medical College, Nashville, Tenn., 1921; died in Billings Memorial Hospital September 15, aged 50, of cerebral vascular accident and hypertension.

Barton Kathcart Thomas ✠ Pottstown, Pa.; Jefferson Medical College of Philadelphia, 1913; affiliated with the Pottstown Hospital; died September 18, aged 62, of cerebral hemorrhage.

Herman H. Thym ✠ Kansas City, Mo.; Kansas City Hahnemann Medical College, 1910; on the staff of Trinity Lutheran and St. Joseph hospitals; died September 12, aged 61, of coronary disease.

G. W. Tootle, Glennville, Ga.; Atlanta Medical College, 1895; member of the American Medical Association; died August 15, aged 79, of angina pectoris.

Henry Christian Viereck, West Orange, N. J.; Medico-Chirurgical College of Philadelphia, 1916; died September 8, aged 57, of coronary embolism and cerebral sclerosis.

John Eliott Wallace, Belmond, Iowa; Bellevue Hospital Medical College, New York, 1890; for many years city physician; died in Steele Memorial Hospital September 6, aged 96, of senility.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Nov. 9, 1949.

National Health Service

National health service is still a main topic of discussion. Interest in this has been greatly stimulated by the arrival of President Truman's delegation and by the subsequent visit of officials of the American Medical Association who were representing this body at the World Medical Association General Assembly held this year in London.

The former group, 16 to 18 in number, was shepherded by the Ministry of Health. It did not make a contact with the Fellowship for Freedom in Medicine, but Lord Horder, its chairman, gave a private interview to five of the members of the group and told them some of the "snags" of the national health service.

The American Medical Association representatives, including ex-President Dr. Sensenich, President-elect, Dr. Henderson, Chairman of the Board of Trustees, Dr. Louis Bauer, and the Editor of THE JOURNAL and editor in chief of the *World Medical Association Bulletin*, Dr. Morris Fishbein, were entertained at dinner by Mr. Laurence Abel, the officers of the fellowship also being Mr. Abel's guests. The American visitors were told the history of the Fellowship for Freedom in Medicine and its aims and objects. They also heard some of the defects of the new service from rural and urban practitioners and also from specialists and consultants.

Lord Horder emphasized the distinction between "socialized" and "nationalized" medicine, a point to which he had referred in his Root Lecture (J. A. M. A. **140**: 1142 [Aug. 6] 1949). If by socialized medicine was meant making medicine available to every citizen, the challenge should be accepted. If it meant passing enactments which put the state in control of medicine, both institutional and domiciliary, so that the doctors became, in effect, whole time paid servants of the state, it should be resisted. The American Medical Association delegates reaffirmed, once more, the determination of the profession in the United States to oppose wholeheartedly this latter attempt.

The present serious fiscal position in the United Kingdom has caused much criticism of the reckless expenditure which signalized the launching of national health service. The initial budget was exceeded by over 40 per cent during the first nine months, calling for a supplementary estimate in the House of Commons of no less than £58,000,000. The Minister of Health explained that to exceed his budget so greatly showed how successful the new service was, a *non sequitur* which caused little or no comment. In view of the present mood of the country the Minister seems to be hesitant about asking for a second supplementary estimate of £16,000,000, which, according to the British Medical Association estimate, he still owes the doctors for work already done.

The Minister of Health stated publicly that, whatever economies the country might be made to effect, "the health service is sacrosanct." A fortnight later the Prime Minister announced that among his suggested "cuts" there was to be a shilling charged on every prescription (formerly dispensed "free") presented to the chemist on national health service forms. The rather specious argument was advanced that this would relieve the pressure on the doctors by the crowds of people attending their "surgeries" and bent on getting something in return for their insurance payment. The Pharmaceutical Society has pointed out in a letter to the Minister that the charge would not deter those who were misusing the service to a serious extent but would be prejudicial to the use of the service by persons who really need medical care.

Among the committees set up by the Minister of Health was a "Distinction Awards Committee," with the president of the Royal College of Physicians as chairman and a personnel of thirteen members whose names suggest that an effort was made to enable them to take as objective a view of the merits of their practicing colleagues as possible. Despite this, however, the work of the committee, and its very existence, has caused a good deal of bitter comment.

The history of the origin and purposes of the Distinction Awards Committee is interesting. The Spens Committee set up to advise the Minister on terms of remuneration of specialists "produced a new idea," says *The Lancet* (Oct. 22, 1949) "for countering the deadening effect . . . of payment according to rank and seniority."

"We are satisfied," the Spens Committee said, "that there is a far greater diversity of ability and effort among specialists than admits of remuneration by some simple scale applicable to all. If the recruitment and status of specialist practice are to be maintained, specialists must be able to feel that more than ordinary ability and effort receive an adequate reward. Moreover, a reward which would be appropriate when these exist would be extravagant when they did not. In consequence we are clear that any satisfactory system of remuneration must involve differentiation dependent on professional distinction." When a specialist has reached his maximum basic salary of £2,500, his pay should cease to depend on length of service; it is suggested that distinction awards, of three different grades, be made to 34 per cent of specialists selected as exceptionally meritorious.

The first move (after a full questionnaire had been sent to every specialist) on the part of this committee has led to criticism: the secretary wrote to a certain key person on the staff of each teaching hospital, such as the dean, suggesting that he ask two or three other members of the staff to discuss with him the relative merits of their colleagues with a view to nominating them for special awards. To those teachers, consultants and specialists who objected to this procedure the retort has been made: "Well, here's a million pounds or so going begging, why not indent for it?"

The most trenchant criticism was probably that of Dr. F. M. R. Walshe (*Brit. M. J.* **2**: 383 [Aug. 13] 1949). "It is one thing," said Dr. Walshe, "to grade a series of posts but quite another to seek the grade professional distinction, and it passes my comprehension that anyone who has spent his working life in medicine—unless as an administrator—could suppose that a grading of this order can be rationally and equitably accomplished. . . . What constitutes 'distinction' in medicine? Is it aptitude in the variegated arts of worldly success? Is it clinical wisdom, therapeutic fervour, or the acceptable usage of the forms of sickroom politeness? Is it the gift of training students or fruitfulness in original research? Is it the acquisition of seniority by lapse of years or a flair for planning the activities of one's colleagues? Is it to be a Mary or a Martha in medicine? How are these incommensurables, these diverse qualities of mind, heart and worldly wisdom, to be comparatively assessed in terms of sterling?"

THE NETHERLANDS

(From Our Regular Correspondent)

Oct. 20, 1949.

Social Medicine

Since the retirement of the professor of bacteriology and hygiene in the Amsterdam University, there has been some rumor that his successor has been appointed for teaching only bacteriology and epidemiology. As the curatorium of the university is considering a chair for social medicine, the vacancy in the teaching of public health (including vital statistics, sanitary organization, occupational hygiene and social insurance) has not yet been filled.

As in other countries there is some disagreement about the definition of social medicine and about the borderline between social medicine and hygiene. Professor Julius (Utrecht) said that social medicine, in the sense in which it is propagated in Holland, is only a hotchpotch. In his opinion all the scientific articles published by the *Netherlands Journal of Social Medicine* are related to epidemiology, to immunology or to vitaminology. Giving a vaccine, a Schick test or a vitamin tablet to a number of children instead of to one child does not create a new science, to be called social medicine. Professor Julius does not deny that there are interesting sociologic problems connected with medicine.

From the teaching point of view this criticism is important. Professors with routine knowledge about laws, organization and registration without scientific background are welcome neither to the faculties nor to the students. A student who obtains clear insight into the etiology and the spreading of tuberculosis or venereal diseases develops a logical interest in the epidemiologic and preventive work of clinics and in other activities cooperating in the campaigns against these diseases. On the other hand, the student will try to escape from lectures on organization, laws and statistics only; such, at least, has been the experience in Holland.

Scrub Typhus in Batavia

In 1909 scrub typhus, caused by Rickettsia orientalis, was recognized among the coolies of the tobacco plantations on Sumatra and described as pseudotyphoid. In later years, after the discovery of the role of Trombicula deliensis, it was called Sumatran mite fever.

Scrub typhus belongs also to the medical history of World War II. Epidemics of scrub typhus occurred among the allied forces in New Guinea. In the last few years cases of scrub typhus have occurred in Batavia, and an investigation was begun by R. Gispen, A. M. Smit and C. D. Westermann (*Med. maandbl.* 2: 238 [Aug.] 1949) in the Queen Wilhelmina Institute of Hygiene of the University and the Eijkman-Institute. In about 1,600 serum tests for Proteus anindolgenes OX (Kingsbury) they found 20 patients whose titer was above 1:800; the history and clinical data for 15 of these were consistent with a diagnosis of scrub typhus. Of these 15 patients, 13 had walked or played on a rather small field of alang grass along the "bandjir" canal of Batavia. Rural sources of infection could be excluded, in view of the special circumstances which prevented civilians from leaving the town. Moreover, rats with Trombiculidae larvae were caught in and near houses on the border of the alang. The disease was observed only during the rainy season and mostly among young persons, the occurrence of scrub typhus in Batavia being more closely connected with recreation than with occupation.

Dr. Fishbein at a Meeting of Excerpta Medica

On October 19 Professor Woerdeman, chief editor of *Excerpta Medica*, presided at a meeting in Amsterdam of the editors and assistant editors of the fifteen sections of the Dutch abstract journal, written in English, which since the last war has developed into an immense enterprise with 5,000 cooperating specialists all over the world.

Dr. Morris Fishbein attended this meeting in his function of foreign chief editor of *Excerpta Medica* and gave an interesting talk on the technical and scientific points connected with the production of good abstracts. At dinner his Dutch confrère of the *Nederlandsch Tijdschrift voor Geneeskunde* expressed his admiration for THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION and in the name of the medical profession thanked Dr. Fishbein for the high standard of this periodical. Before leaving, Dr. Fishbein held a press conference and, according to the *Nieuwe Rotterdamse Courant*, "took the journalists to every part of the medical universe."

ARGENTINA

(From Our Regular Correspondent)

BUENOS AIRES, Oct. 27, 1949.

Institutes of Cardiology

The national Ministry of Public Health on Oct. 4, 1949 inaugurated a Central Institute of Cardiology. The most important center of cardiologic research in the country is the Centro de Investigaciones Cardiológicas under the direction of Dr. Alberto C. Taquini, where a two year course earns postgraduates a diploma in cardiology, granted by the University of Buenos Aires. Cardiologic departments and wards have been inaugurated in a large number of municipal hospitals in Buenos Aires.

Congress of Obstetrics and Gynecology

The seventh Congress of Obstetrics and Gynecology was held in Buenos Aires, October 23-28. The most important subjects were "Syphilis in Pregnancy," "Command and Conduction of Labor," "Genital Tuberculosis" and "Endometrial Cancer." Besides the Argentine physicians the congress was attended by a large number of outstanding specialists from Colombia, Brazil, Chile, Paraguay, Perú and Uruguay.

Drs. H. Alvarez and A. Caldeyro Barcia (Montevideo, Uruguay) made an interesting contribution. The authors recorded the uterine contractions by means of a needle inserted through the abdominal wall into the amniotic liquid. The needle was connected to a manometer, and they observed the existence of uterine contractions during the whole pregnancy. After delivery of the fetus the manometer was connected to the placenta by way of the umbilical vein, and intense contractions were observed, although the woman suffered no pain.

Congress of Anesthesiology

The first Latin American Congress of Anesthesiology was held in Buenos Aires October 17-21. "Anesthesia in Thoracic Surgery" was the subject of the first meeting. The congress was attended by Argentine physicians and delegates from Canada, the United States, Chile, Cuba, Portugal and Venezuela.

Deaths

Dr. Julio Lacoste, former professor of histology of the Faculty of Medicine of Buenos Aires, died recently, aged 68.

Dr. César Ameghino, former professor of psychiatry of the Faculty of Medicine of Buenos Aires, died recently, aged 69.

Correspondence

INFECTION THROUGH MUCOUS MEMBRANE, COLD AND HYALURONIDASE

To the Editor:—In THE JOURNAL (July 30) there was comment on the excellent work of M. L. Som and his co-workers (*Proc. Soc. Exper. Biol. & Med.* **70:** 96, 1949) on the absorption of penicillin through the nasal mucous membrane when hyaluronidase is concurrently administered. The theme is interesting, and deductions of therapeutic interest are evident for sinusitis.

We have under investigation the effects of hyaluronidase when used to facilitate penetration through the nasal mucosa. In the course of our studies on the gentisic acid antihyaluronidase action, we observed that temperature influenced the diffusion power of hyaluronidase in the skin of the rabbit. We then found that if a rabbit is given an intradermal injection with a dye and hyaluronidase, with the Duran-Reynals technic, a stain of the dye is produced that ten minutes later has reached a determined area. If the test is repeated after submission of the region of the skin to be injected to contact with an ice bag and this contact is continued for ten minutes after the injection, the diffusion area of the dye is much larger than in the first instance, the increase being about 50 to 70 per cent (Puig Muset, P.; Salvá, J. A., and G.-Valdecasas, F.: *Med. clin., Barcelona,* to be published).

Our interpretation of this observation is based on the observation that the ischemia produced by cold causes in the tissue an absence of the antihyaluronidase substances (as anti-invasives) which, according to L. W. Wattenberg and F. Glick (*J. Biol. Chem.* **179:** 1213, 1949), are present in the serum but not in the tissues.

The first deduction that we draw from our observation is that it allows us to comprehend better the increase of influenza type infections during winter. The virus that penetrates through the nasal mucous membrane does so because of the hyaluronidase of Coccus type germ (Pneumococcus, Streptococcus) present in mucus, as the virus itself lacks this enzyme. If cold produces an increase in the diffusion power of hyaluronidase, it is easy to understand that the virus or coccus can penetrate with greater ease through the nasal mucosa.

After the publications of Meyer and Ragan (*Science* **108:** 281, 1948) on the antihyaluronidase action of gentisic acid, we studied the possible irritant or necrotic action of this acid. Experiments were performed on the conjunctiva of the rabbit and on its skin with gentisic acid and with salicylic acid. We used the solution in propylene glycol for the ocular application and a 10 per cent preparation in collodion for the cutaneous application. The results showed the known irritant action of salycilic acid and an absolute lack of this property for gentisic acid. A 1 per cent solution of gentisic acid in nasal instillation is now being tested for prophylactic and curative properties in the common cold and influenza. The information we have offers promise. Furthermore, we are trying to diminish infection through the nasal mucosa by utilizing products with antihyaluronidase action, such as gentisic acid.

P. PUIG MUSET,
J. A. SALVÁ and
F. G.-VALDECASAS,
Barcelona, Spain.

AUREOMYCIN AND ALUMINUM HYDROXIDE

To the Editor:—Aureomycin given orally is effective in many microbial infections. The simultaneous oral administration of aluminum hydroxide gel and aureomycin will in some instances reduce the epigastric distress, nausea and vomiting that occasionally occur when aureomycin is given alone. A general inquiry indicates that it is a common practice to prescribe aluminum hydroxide gel with aureomycin. F. E. Di Gangi and C. H. Rogers of the College of Pharmacy of the University of Minnesota suggested that in vitro a suspension of aluminum hydroxide as an adsorbing agent might inactivate aureomycin. It was found that a solution of aureomycin that had been treated with aluminum hydroxide had little antibacterial activity. This prompted us to determine serum levels of aureomycin on 11 human subjects prior to and after they were given aluminum hydroxide gel with aureomycin. Ten of the subjects showed a decided drop in aureomycin serum levels within twenty-four hours after they were given 2 tablespoonfuls of aluminum hydroxide gel with each 0.5 Gm. dose of aureomycin. These observations, which are being published in detail elsewhere, demonstrate that the aluminum hydroxide gel interferes with absorption of aureomycin in the gut and may in certain infections lower the amount of aureomycin in the serum and tissues below an effective therapeutic level. Aluminum hydroxide gel should not be administered with aureomycin.

BURTON A. WAISBREN, M.D.
JEAN S. HUECKEL, Minneapolis.

To the Editor:—In answer to the first question on page 748 in the November 5 issue of THE JOURNAL, whether colloidal aluminum hydroxide diminishes absorption or the efficacy of aureomycin, I would like to make the following comment. According to the information available to us there is a considerable amount of evidence that aluminum hydroxide gel does decrease the absorption of aureomycin and may interfere with its therapeutic effectiveness. This information, incidentally, is so new that I am sure it would not have been available to you at the time the question was prepared for printing.

I trust this information will be of some assistance with regard to this problem.

RUTLEDGE W. HOWARD, M.D.,
Director of Professional Service,
Lederle Laboratories Division,
American Cyanamid Company.

To the Editor:—One of the commonest difficulties in the administration of aureomycin in effective doses is the frequency of nausea and vomiting following gastric irritation. The concomitant administration of aluminum hydroxide and similar slow-acting antacids seems to prevent or relieve the gastric symptoms in some instances but not in others. I have used dinenhydrinate (dramamine) in doses of 100 mg. administered one to one-half hour before the dose of aureomycin, which is taken together with aluminum hydroxide gel or tablets, in 6 patients who had previously shown a severe intolerance to aureomycin with intense nausea and vomiting. These annoying symptoms cleared up immediately, and the patients were able to tolerate up to 1,000 mg. of aureomycin by mouth every four hours without discomfort. As with other antihistaminic drugs some drowsiness was present, which the patient could control by drinking coffee.

While 6 cases do not constitute basis for an authoritative statement, I feel that the distinct relief afforded these patients warrants publication of a preliminary report.

CARLOS P. LAMAR, M.D., Miami, Fla.

Council on Medical Education and Hospitals

UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE FULLY APPROVED

At its meeting on Oct. 22, 1949, the Council on Medical Education and Hospitals voted to extend its approval to the University of Washington School of Medicine. The action of the Council was based on a survey carried out by the Secretary of the Council and a representative of the Association of American Medical Colleges, Oct. 3-11, 1949. This medical school was developed in 1945, and the first students were admitted to the freshman class in the fall of 1946. In accordance with long-standing policy, however, approval is not granted any medical school until representatives of the Council survey the school after instruction has been instituted in all four years of the medical course. The first senior class was enrolled in the fall of 1949 and will graduate in June 1950. The Council's approval on October 22 applies to all students in good standing at the school at that time.

UNIVERSITY OF SOUTH DAKOTA SCHOOL OF MEDICAL SCIENCES FULLY APPROVED

At its meeting on Oct. 22, 1949, the Council voted to remove the University of South Dakota School of Medical Sciences from probation and to restore the school to a status of full approval. This action was based on a detailed survey of the school carried out by the Secretary of the Council and a representative of the Association of American Medical Colleges, Sept. 28-30, 1949. This survey revealed that the deficiencies that were observed which led to the school's being placed on probation have been satisfactorily corrected.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS. *Part III.* Boston, Chicago and New York, January. *Parts I and II,* Feb. 13-15. Centers where there are approved medical schools and five or more candidates. Exec. Sec., Mr. E. S. Elwood, 225 S. 15th Street, Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: *Written.* Various locations. July 21. *Oral.* Philadelphia, April 23-27, Chicago, October 8-12. Sec., Dr. Curtiss B. Hickcox, 745 Fifth Ave., New York 22.

AMERICAN BOARD OF INTERNAL MEDICINE: *Oral.* Chicago, Feb. 8-10. Boston, April 13-15. San Francisco, June 21-23. The oral examinations in the subspecialties will be held at the same time and places. Final date for filing applications for all examinations is Jan. 1. Asst. Sec., Dr. William A. Werrell, 1 West Main Street, Madison 3, Wis.

AMERICAN BOARD OF NEUROLOGICAL SURGERY: *Oral.* Chicago, June 3. Final date for filing applications is Jan. 1. Sec., Dr. W. J. German, 789 Howard Ave., New Haven, Conn.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC. *Written and Reviews of Case Histories. Part I.* Various Centers. Feb. 3. *Oral Part II.* Atlantic City, May 21-28. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh.

AMERICAN BOARD OF OPHTHALMOLOGY: Boston, April. Sec. Dr. Edwin B. Dunphy, 56 Ivie Rd., Cape Cottage, Maine.

AMERICAN BOARD OF ORTHOPAEDIC SURGERY. *Part II,* New York City, Feb. 9-10. Sec. Treas., Dr. Harold A. Sofield, Room 1856, 122 S. Michigan Ave., Chicago.

AMERICAN BOARD OF PLASTIC SURGERY: Examinations are given in June and November of each year in the home town of applicants. Sec.-Treas., Dr. Louis T. Byars, 400 Metropolitan Bldg., St. Louis, Mo.

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY: Spring Examination. Date and location of examination to be announced later. Final date for filing applications is Feb. 1. Sec., Dr. F. J. Braceland, 102-110 Second Ave. S.W.,, Rochester, Minnesota.

AMERICAN BOARD OF UROLOGY: *Written.* Various Centers, Dec. 3. *Oral and Clinical.* Chicago, Feb. 11-15. Sec., Dr. Harry Culver, 7935 Sunnyside Road, Minneapolis 21.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 27-29. Sec., Dr. D. G. Gill, 519 Dexter Avenue, Montgomery.

ALASKA: Juneau, March 7. Sec., Dr. W. M. Whitehead, Box 140, Juneau.

COLORADO: * Denver, Jan. 3-6. Sec., Dr. George H. Gillen, 831 Republic Building, Denver.

DELAWARE: *Examination.* Dover, Jan. 10-12. *Reciprocity.* Dover, Jan. 19. Sec., Dr. J. S. McDaniel, 229 State Street, Dover.

FLORIDA: * *Examination.* Jacksonville, Nov. 27-29. Sec., Dr. Frank D. Gray, 12 N. Rosalind Ave., Orlando.

GUAM: *Endorsement.* Agana, last Friday of each month. Sec., Capt. C. K. Youngkin, Dept. of Public Health, Guam, % F.P.O., San Francisco.

HAWAII: *Examination.* Honolulu, Jan. 9-12. Sec., Dr. I. L. Tilden, 881 S. Hotel St., Honolulu.

IDAHO: Boise, Jan. 9. Exec. Sec., Mr. Armand L. Bird, 305 Sun Building, Boise.

INDIANA: *Examination.* Indianapolis, June. Sec., Dr. Paul R. Tindall, 1138 K. of P. Bldg., Indianapolis.

IOWA: * *Examination.* Des Moines, Dec. 5-7. *Reciprocity.* Des Moines, Nov. 7, Dec. 5. Sec., Dr. M. A. Royal, 506 Fleming Bldg., Des Moines.

KANSAS: Topeka, Dec. 8-9. Sec., Dr. J. F. Hassig, 905 N. Seventh St., Kansas City.

KENTUCKY: Louisville, Dec. 12-14. Sec., Dr. Bruce Underwood, 620 South Third Street, Louisville 2.

LOUISIANA: New Orleans, Dec. 8-10. Sec., Dr. Roy B. Harrison, 1507 Hibernia Bank Building, New Orleans 12.

MARYLAND: *Examination.* Baltimore, Dec. 13-16. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore 1. *Homeopathic. Examination.* Baltimore, Dec. 13-14. Sec., Dr. John A. Evans, 612 West 40th St., Baltimore.

MISSISSIPPI: *Reciprocity.* Jackson, December. Sec., Dr. Felix J. Underwood, State Board of Health, Jackson 113.

NEW HAMPSHIRE: Concord, March 8-9. Sec., Dr. John S. Wheeler, 107 State House, Concord.

NORTH CAROLINA: *Endorsement.* Raleigh, Jan. 23. Sec., Dr. Ivan Procter, 226 Hillsboro St., Raleigh.

NORTH DAKOTA: *Examination.* Grand Forks, Jan. 4-6. *Reciprocity.* Jan. 7. Sec., Dr. C. J. Glaspel, Grafton.

OHIO: *Examination.* Columbus, Dec. 12-14. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: * *Examination.* Oklahoma City, June 7-8. Sec., Dr. Clinton Gallaher, 813 Braniff Building, Oklahoma City.

OREGON: * *Examination.* Portland, January. Exec. Sec., Mr. Howard I. Bobbitt, 608 Failing Building, Portland 4.

PENNSYLVANIA: *Examination.* Philadelphia or Harrisburg, January. Acting Sec., Mrs. Marguerite G. Steiner, 351 Education Building, Harrisburg.

PUERTO RICO: *Examination.* Santurce, March 7. Sec., Mr. Luis Cueto Coll, Box 3717, Santurce.

SOUTH DAKOTA: * Sioux Falls, Jan. 17. Sec., Dr. C. E. Sherwood, 300 First National Bank Building, Sioux Falls.

TEXAS: Fort Worth, Dec. 8-10. Sec., Dr. M. H. Crabb, 1714 Medical Arts Building, Fort Worth.

UTAH. *Examination.* Salt Lake City, June. Dir., Dr. Frank E. Lees, 324 State Capitol Building, Salt Lake City.

VIRGINIA: *Examination.* Richmond, Dec. 2-3. *Reciprocity.* Richmond, Dec. 1. Sec., Dr. K. D. Graves, 631 First St., S.W., Roanoke.

WASHINGTON: * Seattle, January. Director, Department of Licenses, Mr. Edward C. Dohm, Olympia.

WISCONSIN: * *Examination.* Madison, Jan. 10-12. Sec., Dr. C. A. Dawson, River Falls.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ARIZONA: *Examination.* Tucson, Dec. 20. Sec., Mr. Francis A. Roy. Science Hall, University of Arizona, Tucson.

COLORADO: *Examination.* Denver, Dec. 7-8. Sec., Dr. Esther B. Starks, 1459 Ogden Street, Denver.

FLORIDA: *Examination.* June 3. Sec., Mr. M. W. Emmel, University of Florida, Gainesville.

OKLAHOMA: *Examination.* Oklahoma City, April 11. Sec., Dr. Clinton Gallaher, 813 Braniff Building, Oklahoma City.

OREGON: Portland, Dec. 3. Sec., Mr. Charles D. Byrne, State Board of Higher Education, Eugene.

SOUTH DAKOTA: Vermillion, Dec. 2-3. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

TENNESSEE: *Examination.* Memphis, Dec. 30-31. Sec., Dr. O. W. Hyman, 874 Union Avenue, Memphis 3.

WASHINGTON: Seattle, January. Sec., Department of Licenses, Mr. Edward C. Dohm, Olympia.

WISCONSIN: Milwaukee, Dec. 3. Madison, April 1. Sec., Prof. W. H. Barber, Ripon College, Ripon.

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Workmen's Compensation Acts: Ruptured Aneurysm Due to Emotional Upset Following Accident.—This was a proceeding to recover compensation for the death of the plaintiff's husband because of an injury sustained during the course of his employment. From a judgment granting an award, the employer appealed to the Supreme Court of Tennessee.

The deceased, a strong man and in apparent good health until within an hour of his death, severely smashed the last joint of his left ring finger during the course of his employment. After receiving first aid he was driven by a fellow employee to the office of the company doctor, arriving there about twenty-five minutes after the accident had occurred. The doctor administered procaine hydrochloride (novocain®), amputated a part of the finger and dressed the wound all in the course of probably twenty minutes after the patient's arrival. The deceased and his driver then went to his car and started back to his place of business. Between five and ten minutes afterward, and while so returning, the deceased slumped and died almost instantly. This was approximately fifty-five minutes after his finger had been injured.

An autopsy was performed, and it was discovered that he had died of a ruptured aneurysm of the aorta, the artery that leads from the heart. The tear was "about an inch and a half long. It was a jagged tear, it was clean through it and this tear had ruptured directly into the sac around the heart." This condition had been caused by syphilis eating into the walls of the aorta, thus causing a thinning and weakening of the inner parts of the wall. This condition had existed for several years. A person in that condition may die at any time and will die suddenly "if it ruptures into the heart like this one did." Death may come during sleep. The disease is always fatal.

It was the opinion of the doctor who performed the autopsy that the injury to the finger had nothing to do with the death, and that its occurrence about fifty-five minutes before his death was coincidental. The employer insisted that there was no evidence that the injury to the finger in any way contributed to the employee's death. The plaintiff, however, insisted that the accident and its pain excited her husband and that he was excited also by watching the doctor perform the amputation, that this excitement raised his blood pressure and that the increased blood pressure caused the aorta, already weakened by disease, to rupture.

The employer contended that in order to reach such a conclusion it was necessary to base such finding on presumption drawn from presumption as follows: (1) that the employee became excited, (2) that the excitement increased his blood pressure, (3) that the increased blood pressure continued until his death and (4) that the increased blood pressure caused the rupture. It was established by direct testimony of eye witnesses that the deceased manifested emotion from the time he received the injury until he entered the doctor's office and when he left the doctor's office a few minutes before his death. Dr. Wood, whose general qualifications as a doctor were admitted, testified, "In my opinion, any emotion, such as fear and anger or excitement, would tend to raise the blood pressure. The blood pressure would be increased in all persons, in my opinion, but a person with aortic regurgitation would be more susceptible to emotion than the normal person would. . . . The blood pressure increases and causes a rupture of the aneurysm as a result of that." There further appear during his examination the following question and answer: "Now, Doctor, in this individual who had apparently an aortic insufficiency with a concomitant aneurysm, if he had a sudden smashing blow to his finger at 2:30 in the afternoon, could this, in your opinion, cause his blood pressure to raise so rapidly as to put such force on that aortic aneurysm as to cause a lesion in it? A. Yes sir."

We must conclude, said the court, that the foregoing testimony of two fellow employees and the doctor is material evidence in support of the finding of the trial judge that "the inference to be drawn from the facts of this case is that the heightened blood pressure caused by the painful finger injury split the already weakened aorta and hastened the employee's death." The rapid succession of events during the immediately important fifty-five minutes here involved and the sequence in which these events occurred, considered, too, in the light of natural conclusions which result in back tracking from effect to locate cause, seem to make the finding of the trial judge logical. However that may be, said the Supreme Court, those findings are supported by material evidence. That evidence is not made up of mere presumptions, one predicated on the other. It is the direct testimony of eye witnesses as to physical acts and appearances and the testimony of experts as to the possible and probable results of those facts. The result which the expert said was possible and probable from those facts is the result which did obtain here. Accordingly, the judgment of the trial court awarding compensation to the plaintiff for the death of her husband was affirmed.—*Lucey Boiler & Manufacturing Corporation v. Hicks, 222 S.W. (2d) 19 (Tenn., 1949).*

Medical Motion Pictures

FILM REVIEWS

Cancer of the Bladder. 16 mm., color, silent, 350 feet, showing time twelve minutes. Prepared in 1943 by Lowell S. Goin, M.D., and Eugene F. Hoffmann, M.D., Urological and Radiological Departments of Queen of Angels Hospital, Los Angeles. Produced by Billy Burke Productions, Hollywood. Procurable on loan from Lowell S. Goin, M.D., 658 South Westlake, Los Angeles 5.

This is a report type film which demonstrates a new type of therapy, a combination of surgery and low voltage roentgen radiation for the treatment of certain types of cancers of the bladder, mainly those extending into the trigone. One case is followed in detail. The entire procedure is shown from the cardinal symptom of cancer of the bladder, through demonstration of blood in the urine and a preliminary study of an intravenous pyelogram and cystogram to obtain an estimate of the renal function and to demonstrate the extent of the lesion. The diagnosis is then confirmed by direct inspection of the tumor and removal of a section for histologic examination. After performance of the classic suprapubic cystotomy, the necrotic mass of the tumor is removed from its base by electrosurgery and a contact x-ray tube is introduced into the surgical wound. A dose of 7,500 r was given in approximately six minutes to various fields. Microscopic slides are shown before and after radiation therapy. The authors repeat the procedure after an interval of seven to ten days, giving an equal amount of roentgen units to the base of the tumor.

Radiologists, urologists or general physicians interested in the use of contact roentgen radiation in combination with surgical treatment will welcome this motion picture as a good illustration and report of the application of this new method. Since the majority of physicians may not be familiar with the basic principles of this type of radiation, it is useful as part of a lecture on the present status of contact roentgen radiation of accessible lesions. The photography is excellent.

Rehabilitation in Industry. 16 mm., black and white, sound, 1,110 feet, showing time thirty minutes. Produced in 1948 at the General Motors Vauxhall plant in England. Procurable on loan from Dr. Frank R. Ferlaino, Medical Director, General Motors Corporation, 1775 Broadway, New York 19.

This motion picture describes the reconditioning of the human body. It shows in detail how a group of engineers has cooperated with a group of physicians interested in rehabilitation of the disabled to develop in a special retraining shop a large variety of special machines and special industrial tasks which are arranged to provide specific types of corrective exercise. This inspiring film is recommended for industrial physicians, physiatrists and physicians who are concerned with the rehabilitation of seriously disabled persons. The photography, direction and narration are excellent.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1938 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American Heart Journal, St. Louis

37:849-1008 (May) 1949

Observations on Anatomy of Atrioventricular Bundle (Bundle of His) and Question of Other Muscular Atrioventricular Connections in Normal Human Hearts. A. D. Kistin.—p. 849.

Experimental Studies on Validity of Central Terminal of Wilson as Indifferent Reference Point. M. Dolgin, S. Grau and L. N. Katz. —p. 868.

Potassium Autointoxication from Hemolysis of Red Cells. J. Wener, H. Stansfield, H. E. Hoff and H. A. Winter.—p. 881.

Analysis of Relative Accuracies of Wilson and Goldberger Methods for Registering Unipolar and Augmented Unipolar Electrocardiographic Leads. M. B. Rappaport and C. Williams.—p. 892.

Transient Ventricular Fibrillation: II. Effects of Gradually Induced Oxygen Deficiency on Patients with Transient Ventricular Fibrillation and on Patients with Periodic Standstill of Ventricle. S. P. Schwartz, G. C. Leiner and R. J. Michtom.—p. 918.

T Waves Normal in Size and Direction but Abnormal in Contour. V. Alzamora-Castro, C. Rubio, G. Battilana and R. Subiria.—p. 927.

Catheterization of Coronary Sinus in Man. J. W. Culbertson, M. H. Halperin and R. W. Wilkins.—p. 942.

Simple Bipolar Technic for Analysis of Vector Relationship Between Unipolar and Standard Electrocardiographic Leads. N. A. Cohen and A. M. Glicksman.—p. 952.

American Journal of Clinical Pathology, Baltimore

19:501-598 (June) 1949

Experimental Ovarian Tumors in Rats. G. R. Biskind and M. S. Biskind.—p. 501.

Neurogenesis of So-Called Granular Cell Myoblastoma. J. A. Fust and R. P. Custer.—p. 522.

Evaluation of Use of Group-Specific Substances in Group O Blood Transfusions. O. M. Alton and W. R. Platt.—p. 536.

Diffusely Enlarged Uterus. K. M. Truemner and D. H. Kaump.—p. 544.

Observations on Occurrence of Varices of Esophagus in Routine Autopsy Material. T. Weinberg.—p. 554.

19:599-700 (July) 1949

*Medicolegal Examination of Bodies Recovered from Burned Buildings. F. R. Dutra.—p. 599.

Studies on Inclusion Bodies: I. Acid-Fastness of Nuclear Inclusion Bodies That Are Induced by Ingestion of Lead and Bismuth. M. Wachstein. —p. 608.

Lecithinase Activity in Splenic Dyscrasias. G. J. Scheff and A. J. Awny. —p. 615.

*Studies on Blood Factor rh^w. A. S. Wiener and E. B. Gordon.—p. 621.

Medicolegal Examination of Burned Bodies.—According to Dutra, examination of a body recovered from a burned building should reveal whether the person was alive or dead at the onset of the conflagration. If death has occurred in a fire the blood will almost invariably show a high level of carbon monoxide. In addition, particles of soot are usually present on the mucosae of the larynx, trachea and bronchi. The presence of true blisters, or of a peripheral zone of hyperemia, associated with burns of the skin indicate life at the time the burns were sustained. If death occurred in the fire, analysis of the blood for ethyl alcohol may reveal that escape was rendered more difficult by inebriation. Examination of the viscera and extremities will reveal whether injury or disease prevented escape. Certain postmortem artefacts of burned bodies produced by heat may be confused with premortem injuries. These artefacts include splits of the skin which simulate incised wounds, pseudoblisters caused by formation of steam beneath the epidermis, cracks of the outer table of the skull from drying and shrinkage, fractures of the entire thickness of the skull from the generation of steam in the cranial cavity and epidural hematomas over the vertex of the brain. Most deaths in conflagrations are due to heat, noxious gases (especially carbon monoxide) or the combined effects of heat and gases. Death from heat is indicated by extensive burns of the body, together with hemorrhages beneath the endocardium of the septal surface of the left ventricle of the heart. Identification of partially burned bodies may be made from dental peculiarities or from individual physical characteristics, such as congenital abnormalities or residua of disease or surgery.

Blood Factor rh^w.—Wiener and Gordon carried out agglutination tests with blood specimens from 259 white, 104 Negro and 31 Chinese persons classified according to their Rh-Hr blood types. Among the 259 white persons there were 10 whose blood samples were clumped by anti-rh^w serum, but not one specimen of blood of the 104 Negro and 31 Chinese persons was clumped by anti-rh^w serum. All 10 samples clumped by rh^w antiserums contained the factor rh′, indicating that rh^w is a partial antigen associated with factor rh′, especially in bloods containing agglutinogen Rh$_1$, just as factors rh′ and rh″ are partial antigens associated with Rh$_0$ in particular Rh$_0$ positive bloods. These results confirm previous observations indicating the existence of further variants of the Rh factor in addition to the eight standard varieties presumably transmitted by corresponding allelic genes.

American J. Obstetrics and Gynecology, St. Louis

58:1-206 (July) 1949. **Partial Index**

Developmental Pathology: New Field in Medicine. P. Gruenwald.—p. 1.

*X-Ray Visualization of Placenta: Experiences with Soft-Tissue and Cystographic Technics in Diagnosis of Placenta Previa. C. S. Stevenson.—p. 15.

*Saddle Block Anesthesia. H. E. Schmitz, J. E. Towne and G. Baba. —p. 30.

Ten-Year Study of Cesarean Section in Rochester and Monroe County, 1937 to 1946. J. K. Quigley.—p. 41.

Ovogenesis in Adult Human Ovary. O. H. Schwarz, C. C. Young Jr. and J. C. Crouse.—p. 54.

Sickle Cell Anemia and Pregnancy. G. W. Anderson and T. Busby. —p. 75.

Effect of Veratrum Viride on Urine Volume, Blood Pressure and Pulse Rate in Normal and Toxemic Pregnancy. N. S. Assali, R. W. Kistner and S. T. Garber.—p. 90.

Penicillin Therapy in Obstetrical Patient: Study of Its Effect on Bacterial Flora of Postpartum Uterus. J. A. Guilbeau Jr., I. G. Schaub and M. C. Andrews.—p. 101.

Effect of Birth on Mortality. M. Battle.—p. 110.

Diagnosis and Management of Rupture of Uterus with Study of 64 Maternal Deaths. C. A. Gordon and A. H. Rosenthal.—p. 117.

Hydatidiform Mole Followed by Postpartum Eclampsia and Chorionepithelioma, with Recovery. C. W. Mueller and W. A. Lapp.—p. 133.

Multiple Pregnancies at Chicago Lying-In Hospital, 1941 to 1947. E. L. Potter and H. Fuller.—p. 139.

Myomas During and After Menopause. P. R. Zeit.—p. 153.

Clinical Evaluation of Sulfamerazine in Postpartal and Postabortal Sepsis. L. S. Robinson and C. G. Johnson.—p. 166.

Effect of Sulfonamide Cream on Bacterial Flora of Infected Vagina and Cervix. G. Blinick, P. Steinberg and J. V. Merendino.—p. 176.

Pregnancy Subsequent to Radical Mastectomy of Breast for Cancer. E. Steffen and H. Grace.—p. 180.

Roentgen Visualization in Diagnosis of Placenta Previa.—According to Stevenson it is an established fact that the placenta can be roentgenographically visualized in over 90 per cent of all women who are in the third trimester of pregnancy. During the past several years roentgen studies were made at the Boston Lying-in Hospital in all cases of uterine bleeding and of transverse or oblique position of the fetus occurring during the third trimester of pregnancy. Stevenson reports on the roentgen diagnosis of placenta previa with the aid of soft tissue technic introduced by Snow and Powell and the cystographic method first described by Ude, Weum and Urner. Placement of the patient in such fashion that an edgewise view of the central vertical axis of the placenta will be represented on the roentgenogram is of prime importance. It was impossible to visualize the placenta in the roentgenograms of 14 women in a total series of 488 cases. In the 474 cases in which the placenta was visualized, correct diagnosis of the presence or absence of placenta previa was made in 98.1 per cent. There were 39 cases of placenta previa, and all of these were diagnosed correctly except in 3 instances, in which marginal placenta previa was present. This gave a correct diagnostic achievement of 100 per cent in complete previa (21 cases), of 100 per cent in partial previa (8 cases) and of 70 per cent in marginal previa (10 cases). Accuracy in the differential roentgen diagnosis between marginal placenta previa and low-implanted placenta was difficult to attain. No maternal mortality and no increase in fetal or neonatal mortality resulted

from three "false negative" diagnostic errors. Since special equipment is not necessary, the roentgenologic technic is simple; a resident obstetrician or any interested roentgenologist can learn to read the roentgenograms. The method is a useful aid in the diagnosis of the cause of uterine bleeding in the latter weeks of pregnancy.

Saddle Block Anesthesia.—Schmitz and associates analyzed 877 attempts at saddle block anesthesia with heavy dibucaine hydrochloride solution that were carried out at a Chicago maternity hospital during a period of two years. The anesthesia provided complete subjective relief for at least one hour in 816 cases, or 93 per cent, and incomplete subjective relief in 53 cases, or 6 per cent. Seven hundred and fifty-four patients, or 86 per cent, were delivered with the sole analgesic effect of saddle block anesthesia. In 123 other patients another anesthetic was used to supplement waning spinal effect, for deeper anesthesia to permit operative maneuver and for psychologic reasons only. Subjective relief was of one to two hours' duration on the average, but the perineal anesthesia usually exceeded three to four hours' duration. The time of administration was the late first stage or early second stage of labor. The incidence of operative deliveries was low. The untoward effects included motor weakness in all cases, a considerable fall in blood pressure in nearly a sixth of the patients, postspinal headaches in 14 per cent of the cases and spinal reaction and "neurologic complication" (foot drop) in 1 case each. There were 6 stillbirths and 4 neonatal deaths; 2 of the latter were due to prematurity. The authors feel that saddle block anesthesia is the most nearly ideal for obstetric analgesia and anesthesia if used judiciously by an experienced obstetrician. The maximum safety to the fetus and mother, the dramatic relief of the discomfort of labor and the simplicity of the technic make it attractive for the mother, the nursing staff and the attending obstetrician. It must be borne in mind that there are potential dangers to this type of anesthesia.

American Journal of Physiology, Baltimore

157:1-176 (April) 1949. **Partial Index**

Effect of Increased Renal Venous Pressure on Renal Function. W. D. Blake, R. Wégria, R. P. Keating and H. P. Ward.—p. 1.
Reabsorption of Creatine and Guanidoacetic Acid by Renal Tubules. E. A. H. Sims and D. W. Seldin.—p. 14.
Role of Kidney in Pathogenesis of Hypertension as Determined by Study of Effects of Bilateral Nephrectomy and Other Experimental Procedures on Blood Pressure of Dog. A. Grollman, E. E. Muirhead and J. Vanatta.—p. 21.
Effects of Emotional Disturbance on Water Diuresis and Renal Blood Flow in Rabbit. J. Brod and J. H. Sirota.—p. 31.
Enhancing Effects of Growth Hormone on Renal Function. H. L. White, P. Heinbecker and D. Rolf.—p. 47.
Comparison of Carbohydrate Effects of Adrenalin Infused into Femoral Vein, Carotid Artery, Aorta and Portal Vein of Rats. S. Sherlock. —p. 52.
*Factors in Explosive Decompression Injury. E. L. Corey.—p. 88.
Minimal Effective Dose of Intravenously Administered Histamine in Pregnant and Non-Pregnant Human Beings. H. Janowitz and M. I. Grossman.—p. 94.
Analgesia and Anesthesia Induced by Epinephrine. A. Leimdorfer and W. R. T. Metzner.—p. 116.
Measurement of Experimentally Induced Brain Swelling and Shrinkage. K. A. C. Elliott and H. Jasper.—p. 122.
Pulmonary Lesions in Guinea Pigs with Increased Intracranial Pressure, and Effect of Bilateral Cervical Vagotomy. G. S. Campbell and M. B. Visscher.—p. 130.
Studies in Fractionation of Liver: Composition of Regenerating Liver After Partial Hepatectomy in Rats. A. Chanutin and E. C. Gjessing. —p. 135.
Calorie Intakes in Relation to Quantity and Quality of Protein in Diet. D. M. Hegsted and V. K. Haffenreffer.—p. 141.
Effects of Atropine on Food Ingestion and Water Drinking in Dogs. J. W. Archdeacon, M. W. Presnell and C. J. Walton.—p. 149.
Estimation of Relative Velocities of Plasma and Red Cells in Circulation of Man. E. D. Freis, J. R. Stanton and C. P. Emerson.—p. 153.
Tetraethylammonium as Aid in Study of Cardiovascular Reflexes. G. K. Moe, B. R. Rennick, L. R. Capo and M. R. Marshall.—p. 158.
Hemodynamics of Aortic Occlusion. A. Van Harreveld, G. A. Feigen and L. S. Lerman.—p. 168.

Factors in Explosive Decompression Injury.—Corey says that conventional studies on explosive decompression have involved the sudden alteration of the environment from that of the normal, ambient atmosphere to rather high vacuums, in simulation of pressure-cabin failure. In such experiments test animals are simultaneously subjected to: (*a*) the rapid expansion of contained gasses; (*b*) anoxic anoxia, and (*c*) aeroembolism. Qualitative evaluation of the relative importance of these factors in the causation of injury and death from rapid decompression has been difficult. In experiments designed to eliminate the presence of anoxic anoxia, rats were explosively decompressed to normal atmospheric pressure after exposure to positive pressures of from 2 to 30 atmospheres for varying time intervals. These experiments indicate that anoxic anoxia and aeroembolism constitute the major factors in the causation of explosive decompression injury and that intrapulmonary gas expansion may be considered of relatively minor importance as a cause of death.

American Journal of Psychiatry, New York

105:881-960 (June) 1949

Learning Capacity in Schizophrenia with Special Reference to Concept of Deterioration. P. E. Huston and D. Shakow.—p. 881.
Observations on Histopathology of Schizophrenia: I. Cortex. N. W. Winkelman and M. H. Book.—p. 889.
Brain Tumors in Mental Hospital Patients. S. Levin.—p. 897.
Spanish Psychiatry During Last Decade. G. R. Lafora.—p. 901.
Industrial Psychiatric Program in Government Setting. L. D. Ozrin and L. M. Herrick.—p. 904.
Addiction Liability of Methadon (Amidone, Dolophine, 10820) and Its Use in Treatment of Morphine Abstinence Syndrome. H. Isbell and V. H. Vogel.—p. 909.
Flight from Insight. A. B. Wheelis.—p. 915.

American Journal of Public Health, New York

39:839-968 (July) 1949. **Partial Index**

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Child Health Institute in Rochester, Minnesota. B. Spock.—p. 854.
Relation of Nutrition to Infection in Children. J. M. Rawlings.—p. 858.
Influence of Protein Nutrition on Experimental Infection: Physiologic Aspects. J. Metcoff.—p. 862.
Laboratory Tests in Diagnosis of Brucellosis. N. B. McCullough.—p. 866.
Chronic Brucellosis: Unsatisfactory Status of Current Diagnostic Methods. H. J. Harris.—p. 870.
*Rabies Vaccine Encephalomyelitis in Relation to Incidence of Animal Rabies in Los Angeles. C. F. Pait and H. E. Pearson.—p. 875.
Progress Report on Hospital Survey and Construction Act. V. M. Hoge. —p. 889.
Quality of Medical Care in National Health Program. D. A. Clark and others.—p. 898.

Rabies Vaccine Encephalomyelitis.—Pait and Pearson state that in Los Angeles City and County approximately 200 rabid dogs are detected annually. There is only 1 human case per year, but at least 800 persons are given rabies vaccine. The chance of acquiring rabies from known dog bites in Los Angeles is estimated to vary from 1:1,400 to 1:2,100. There were 9 cases, 1 fatal, of postvaccinal encephalomyelitis among 5,500 treated persons, an incidence of 1:600. The data presented here and elsewhere make it apparent that the incidence of rabies vaccine encephalitis is a real contraindication to its indiscriminate use. It is likely that the incidence will be reduced when a more completely purified product of brain tissue is produced; at present most vaccines consist of approximately 10 per cent suspensions of rabbit brain. There is evidence that human rabies can be largely prevented by controlling the disease in dogs. There is a large and highly vocal group of dog fanciers who tend to resist all efforts to interfere with the dog population. While such persons may reject programs based on protection of human beings, they can scarcely fail to accept one designed primarily for the protection of dogs.

American Journal of Surgery, New York

77:679-800 (June) 1949

Modern Management of Acute Perforated Gastroduodenal Ulcers. D. C. Collins.—p. 684.
Carcinoma of Breast: Present Status of Therapy. H. W. Jones Jr. —p. 696.
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Fat Embolism. J. E. Dunphy and F. W. Ilfeld.—p. 737.
Acute Appendicitis in Infants and Children Under Ten Years of Age. J. J. Greene.—p. 744.
Carcinoma of Colon Producing Acute Intestinal Obstruction During Pregnancy. P. S. Putzki, J. H. Scully, J. Kotz and others.—p. 749.
Hysterogram in Diagnosis of Uterine Bleeding. D. J. Wexler, C. H. Birnberg and L. Kurzrok.—p. 755.
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American Practitioner, Philadelphia

3:579-642 (June) 1949

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Clinical Investigation of Tagathen (Chlorothen Citrate) (N,N-dimethyl-N1-(2 pyridyl)-N1-(5-chloro-2 thenyl) ethylene diamine citrate) with Comparison of Various Antihistaminic Agents. S. J. Taub, R. E. Miller and R. G. Taub.—p. 586.
Management of Surgical Chest. R. B. White, E. Glenn, B. J. Tatarowicz and C. Allison.—p. 589.
Problem of Weakness and Fatigue. M. Y. Silver.—p. 598.
Hypertension of Renal Origin with Report of 3 Cases. T. Levy.—p. 604.
Folic Acid and Neurologic Manifestations of Pernicious Anemia. W. M. Fowler and A. B. Hendricks.—p. 609.
Report on Hemophilus Influenzae Meningitis. E. F. P. Kearney.—p. 614.
Triad of Urethritis, Conjunctivitis and Arthritis. A. M. Lefkovits.—p. 619.
Diagnosis and Treatment of Salivary Calculus. A. P. Seltzer.—p. 626.
Infectious Mononucleosis: Report of Unusual Case with Hepatitis Studied by Serial Liver Biopsies and Complicated by Hemolytic Anemia. H. F. Gloyne.—p. 628.

3:643-704 (July) 1949

Recent Advances in Diagnosis and Treatment in Office Gynecology. W. J. Reich, M. J. Nechtow and M. W. Rubenstein.—p. 643.
Urinary Tract Complications in Obstetrics and Gynecology. A. J. Murphy.—p. 647.
*Inhalation of Penicillin Dust; Its Proper Role in Management of Respiratory Infections. L. R. Krasno and P. S. Rhoads.—p. 649.
Cutaneous Manifestations of Systemic Disease. J. G. Downing.—p. 654.
Benadryl and Stramonium in Parkinsonism. H. Pettit.—p. 662.
Allergic Manifestations of Eye and Surrounding Structures. S. J. Taub, M. J. Fowler, B. R. Gutow and others.—p. 664.
Diagnosis of Early Acute Anterior Poliomyelitis. J. A. Luhan.—p. 673.
Care of Feet in Peripheral Vascular Disease. A. Fields.—p. 678.
*Lipemic Nephrosis With and Without Nephritis. W. Heymann and L. J. Alperin.—p. 680.
Mercuhydrin (Meralluride) Suppositories as Diuretic in Congestive Heart Failure. M. Kissin and J. J. Stein.—p. 684.
Few Children Need Die. C. F. McKhann.—p. 688.
Insulin Resistant Diabetes Mellitus Associated with Dwarfism. N. Burnstien and McL. Patterson.—p. 690.
General Medical Practitioner and Plastic Surgery. A. P. Seltzer.—p. 695.

Inhalation of Penicillin Dust in Respiratory Infections.—Krasno and Rhoads employed inhalation of penicillin dust in over 1,000 patients and established the usefulness of this treatment in all bacterial infections of the upper part of the respiratory tract and those of the trachea, bronchi and bronchioles, including bronchiectasis. It is helpful in preventing bacterial complications of the common cold and in controlling infection of the upper part of the respiratory tract which may accompany pneumonia, but it should never be used as the sole bacteriostatic agent in pneumonia to the exclusion of parenterally introduced penicillin or sulfonamide drugs. In some instances penicillin dust inhalation is the only treatment required, but often this treatment should be used in conjunction with one or more of the commonly accepted means of combating infection of the upper part of the respiratory tract, such as vasoconstrictors, bronchodilators, antihistaminics, removal of allergens and surgical drainage. For deep seated infections with severe systemic manifestations, it should be used in conjunction with parenterally introduced penicillin or sulfonamide drugs. Allergic reactions resulting from this method of penicillin administration are, except in rare instances, limited to the mucous membranes of the mouth and throat and—if the need of local penicillin concentrations is great—do not necessarily constitute a contraindication to its continued use. There has been no evidence that penicillin dust creates penicillin fastness in micro-organisms if adequate treatment is given. Patients with bronchiectasis should receive penicillin dust over long periods and in dosage which keeps them symptom free.

Lipemic Nephrosis.—Heymann and Alperin point out that current texts on pediatrics classify cases of nephrosis in which at onset or during later stages some hypertension, azotemia or hematuria are also noted, as chronic glomerulonephritis (nephrotic glomerulonephritis), nephrotic phase of chronic glomerulonephritis, chronic nonspecific nephritis and nephrotic nephritis. These terms suggest that lipemic nephrosis, which is usually accepted as a clinical entity by pediatricians, and nephrosis associated with signs of nephritis are two different entities. Observations reported here are based on 65 cases. In 23 no evidence of nephritis was noted during the entire course of a disease with the characteristic features of lipemic nephrosis. In 42 of these patients signs of an additional inflammatory renal lesion were observed. The analysis of these cases does not support the view that these two conditions represent different disease entities. The degree of lipemia and the age distribution are the same in both forms. The nephrotic diseases have a different age distribution than does acute hemorrhagic glomerulonephritis. The maximum disposition for the nephroses is found in the second year of life, while glomerulonephritis is still rare. The curve for the latter increases with age up to puberty, while the curve for the nephrotic diseases decreases. The observation of preceding infections in patients with acute glomerulonephritis is not noted regularly in children ill with nephrosis with or without nephritis. The view that nephrosis represents one disease entity unrelated to chronic glomerulonephritis finds further support in the fact that nephrosis with and without nephritis has been produced in rats by the intravenous injection of nephrotoxic serums obtained from rabbits. Neither this nor any other method has so far produced acute and chronic glomerulonephritis. The authors prefer the term "lipemic nephrosis with or without nephritis." It keeps the nephrotic diseases in one group, separates them from chronic glomerulonephritis and does not imply the priority of the nephritic component. It also alters the hopeless prognosis for chronic glomerulonephritis which has been observed in nephrosis with nephritis.

Archives of Ophthalmology, Chicago

41:659-780 (June) 1949

*Blood Groups and Effects of Roentgen Irradiation in Retrolental Fibroplasia. T. O. Paul.—p. 659.
*Differential Diagnosis of Boeck's Sarcoidosis: Report of 10 Cases with Ocular Involvement. B. L. Gifford and A. C. Krause.—p. 667.
Ocular Findings in 323 Patients with Schizophrenia: Preliminary Report. M. Cohen.—p. 697.
Reserve Accommodation. H. Eggers.—p. 701.
Supposed Torsion of Eye Around Visual Axis in Oblique Directions of Gaze. M. Márquez.—p. 704.
Neurofibromatosis Associated with Tumors of Optic Papilla: Report of Case. J. Goldsmith.—p. 718.
Contact Lens Electrode for Iontophoresis. P. Tower.—p. 730.
Congenital Cyst of Vitreous: Report of Case. J. V. Cassady.—p. 734.
Metabolism of Cornea. T. D. Duane.—p. 736.

Retrolental Fibroplasia.—According to Paul, retrolental fibroplasia usually occurs bilaterally in premature infants and unilaterally in full term infants. It is characterized by a gray pupillary reflex, retrolental vascular membrane, microphthalmos, shallow anterior chamber with anterior synechias (a feature which predisposes the eye to the development of secondary glaucoma), remains of an anterior pupillary membrane, spheroidal lens and searching nystagmus. The lens is clear. The pupil may or may not be irregular in outline, according to whether or not adhesions have formed. Small strands and remnants of pupillary membrane, like a spider web, can be observed passing across the pupil. The author studied 12 babies with retrolental fibroplasia, all of whom had deep sunken eyes and discoloration of the lids. The blood groups were studied in 6 of them, including the blood group of the parents. The Rh factor of the infants was also determined, but the assumption that a blood factor might be a cause of prematurity in this congenital malformation could not be definitely established. Further studies of this type at several medical centers, with possibly a central registry, might produce useful information. Some of the infants were given roentgen irradiation, the total dose ranging from 400 to 1,000 r, in an attempt to devascularize the membrane. Of 9 eyes treated with roentgen radiation, 3 showed slight improvement, with thinning of the membrane at the less dense borders, and 6 showed no apparent change. In no case was the thinning sufficient to be regarded as of real help preliminary to needling of the lenses. Little or no effect on the vessels of the membrane could be observed. The differential diagnosis of retrolental fibroplasia must take into consideration retinoblastoma, serous detachment of the retina and congenital cataract.

Sarcoidosis with Ocular Involvement.—Gifford and Krause show that the diagnosis of sarcoidosis may be arrived at on the basis of examination of the eyes, biopsies of the skin, lymph nodes and nasal mucosa, and roentgenographic studies of the chest and of the bones of hands and feet. The patient may be seen by the ophthalmologist, the otolaryngologist, the dermatologist, the chest clinician and the roentgenologist, with the

pathologist often giving an opinion on biopsies. Some of the problems of diagnosis pertaining to each specialty are discussed. The authors stress the need of conservativism in the diagnosis of sarcoidosis, pointing out that many cases have been presented as instances of this disease with incomplete evidence. Sarcoidosis is chiefly confused with tuberculosis, and less often with Hodgkin's disease, syphilis, neoplasm, silicosis, leukemia, leprosy and other forms of lymphoblastoma. Sarcoidosis is rare. In the clinics of the University of Chicago only about 30 cases are being followed, and this number has been gleaned from over 450,000 registrations since 1927. The eyes were involved in 10 of the 30 cases. Five of the 10 patients were Negroes. Since patients of this race comprise only about 1 per cent of patients coming to these clinics, it appears that sarcoidosis is many times more frequent in Negroes than in white persons. The usual sequence of events for the ocular disease was the appearance of conjunctivitis followed by keratitis, with anterior uveitis occurring with the conjunctivitis or keratitis, or following at a later period. Secondary glaucoma was not observed; complicated cataract was infrequent, and chorioretinitis was rare. The prognosis for good vision was excellent, even though the anterior uveitis was severe and active for many years. However, this was true only in cases without corneal opacification.

Archives of Otolaryngology, Chicago

48:379-506 (Oct.) 1948

Laryngectomy: Surgical Technic Which Promotes Rapid Healing. J. B. Erich.—p. 379.
Carcinoma of Nasopharynx. C. C. Fox.—p. 390.
Value of Caloric Test of Labyrinth. L. B. W. Jongkees.—p. 402.
*Streptomycin by Topical Application in Treatment of Chronic Suppurative Otitis Media. A. E. Lundon.—p. 418.
Paranasal Sinuses. S. Salinger.—p. 430.
Functional Examination of Hearing. A. Lewy, S. L. Shapiro and N. Leshin.—p. 463.

Topical Application of Streptomycin in Otitis Media.—Lundon discusses topical application of streptomycin in the treatment of chronic suppurative otitis media and reviews histories of 20 patients treated by this method in the outpatient department of a Montreal hospital. The requisites for streptomycin therapy were as follows: (*a*) the existence of chronic suppurative otitis media with well established chronicity; (*b*) sensitivity of the invading organisms to streptomycin, and (*c*) the topical application of penicillin (5,000 units per cubic centimeter) twice daily for two weeks. Of the group of 20 patients whose treatment was completed, 15 were shown to have responded favorably, as evidenced by "dry ears" and absence of pathogenic bacteria in the majority of cases; 5 were regarded as therapeutic failures, 3 of whom gave evidence of some other pathologic condition, probably closely associated with suppuration of the middle ear and involvement of the mastoid. Duration of treatment varied from one to twelve weeks. In 8 of the 15 cases with successful outcome it did not exceed three weeks; for obvious reasons, the estimation of the time factor in connection with outpatients was not found practical. The author believes that the topical application of streptomycin has a definite place in the treatment of chronic suppurative otitis media. It may well be the treatment of choice in many cases in which formerly there has been no response to treatment. There are apparently no contraindications to the topical use of the drug. Local reactions did not occur.

Archives of Pathology, Chicago

48:1-104 (July) 1949

Calcification of Myocardium: Pathologic Study of 13 Cases. I. Gore and W. Arons.—p. 1.
Lipid Fractions of Human Adrenal Glands. E. Adams and M. Baxter.—p. 13.
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Deposition and Fate of Plutonium, Uranium and Their Fission Products Inhaled as Aerosols by Rats and Man. K. G. Scott, D. Axelrod, J. Crowley and J. G. Hamilton.—p. 31.
New Elastic Fibers Formed in Wall of Gallbladder in Disease of Gallbladder: Pathologic, Clinical and Roentgenologic Data Concerning Hitherto Neglected Lesion. J. L. Riopelle.—p. 55.
Pathogenesis of Experimental Hepatic Fibrosis and Cirrhosis in Dog. T. Gillman and I. L. Chaikoff.—p. 67.
Adenoma of Ceruminous Gland in Dog. M. B. Black.—p. 85.
Effect of Experimental Thiamine Deficiency on Heart of Rhesus Monkey. J. F. Rinehart and L. D. Greenberg.—p. 89.

Archives of Pediatrics, New York

66:233-282 (June) 1949

*Dietary Control of Dental Caries. M. S. Cioffari.—p. 233.
Coccidioidomycosis: Case Studies in Children. R. Cohen.—p. 241.
Opinions of Pediatricians on Certain Problems of Infant Care. G. F. Weinfeld.—p. 266.

Dietary Control of Dental Caries.—Cioffari reports on two groups of 50 children whose dental condition was observed regularly from birth until 5 years of age. One group had few sweets in their diet, with additional emphasis on coarseness of food and regular vitamin supplements. The control group had the average American diet with plenty of sweets and irregular vitamin supplements; both groups used the same water supply. At 5 years of age, 78 per cent of the group under diet control had no visible cavities with 0.18 cavity per child, while only 6 per cent of the controls had no cavities with 5.22 cavities per child, a thirteen fold difference. Evidence points to easily fermentable carbohydrates, especially sucrose, as the main offenders. A child's diet, at least until he is 7 to 8 years of age, should include (*a*) a bare minimum of sweets, (*b*) a good amount of coarse foods and (*c*) a maintenance amount of vitamins. In addition, it might be advisable to give the unavoidable quota of sweets at the beginning of the meal only, and to have a raw, coarse fruit or vegetable at the end, with raw fruit for snacks.

Archives of Surgery, Chicago

58:731-926 (June) 1949

*Prefrontal Lobotomy in Management of Intractable Pain. F. E. Hamilton and G. J. Hayes.—p. 731.
Surgical Management of Instrumental Perforation of Esophagus. J. D. Bisgard and H. H. Kerr.—p. 739.
*Hemihepatectomy with Hepaticojejunostomy for Irreparable Defects of Bile Ducts. R. L. Sanders.—p. 752.
Gastrojejunocolic Fistula. S. E. Lawton and A. R. Marks.—p. 762.
Phlegmonous Cecitis: Report of 2 Cases. W. L. Riker, R. K. Gilchrist and V. C. David.——p. 772.
Hidden Carcinoma of Breast. H. D. Cogswell.—p. 780.
Effects on Breast of Removal of Nipple or Severing of Ducts. H. H. Davis.—p. 790.
Mediastinal Tumors. A. R. Curreri and J. W. Gale.—p. 797.
Suppurative Disease of Lungs. F. R. Harper, W. B. Condon and W. H. Wierman.—p. 819.
Hernia Into Umbilical Cord and Omphalocele (Amniocelle) in Newborn. C. D. Benson, G. C. Penberthy and E. J. Hill.—p. 833.
Thumb Traction Technic for Reduction of Colles' Fracture. R. G. Carothers and F. J. Boyd.—p. 848.
Classification and Treatment of Trochanteric Fractures. H. B. Boyd and L. L. Griffing.—p. 853.
Hemipelvectomy for Malignant Tumors of Bony Pelvis and Upper Part of Thigh. R. A. Wise.—p. 867.
Carcinoma of Thyroid Gland. A. S. Jackson.—p. 875.
Elderly Patients as Surgical Risk: Analysis of 322 Operations Performed on 244 Patients Sixty Years of Age and Over. W. H. Parsons and W. K. Purks.—p. 888.
Congenital Anorectal Atresia: Report of Case. R. J. Patton.—p. 907.

Prefrontal Lobotomy in Intractable Pain.—Hamilton and Hayes used the Lyerly technic of prefrontal lobotomy with modification. There were 3 deaths among the 16 patients treated, 1 due to hemorrhage, 1 following convulsion and the last from a self-inflicted infection of the lobotomy incision. The results obtained range from complete denial of pain to admission of no benefit. The intermediate levels are (*a*) affirmative statements of pain on direct questioning but absence of any spontaneous complaint referable to it, (*b*) occasional complaints of pain made spontaneously but with no apparent real distress requiring medication and (*c*) a lowering of the preoperative general distress, with a lessened but continued need for medication. These results have been graded 4 plus for denial of pain to zero for no benefit. With this grading 5 of the 15 surviving patients fell into 4 plus category, 7 into the 3 plus, and one each into the 2 plus, 1 plus and zero category. Although the authors consider lobotomy a useful procedure in certain circumstances, they do not believe that it should be performed indiscriminately.

Hemihepatectomy with Hepaticojejunostomy for Irreparable Defects of Bile Ducts.—Sanders reports a woman, aged 23, who had had a cholecystectomy elsewhere eight months previously. She became jaundiced soon after the operation. The surgeon had inserted a tube for drainage, which was still

in situ and was draining. There were frequent episodes of chills and fever. When the abdomen was opened, the common duct was identified with difficulty because of extensive adhesions. The distal portion was so short that an end to end anastomosis with the proximal portion was impossible. The proximal portion had sloughed away into the hilus of the liver, and a complete stricture in this area had produced a large biliary cyst. After removal of the contents of the cyst, the segment was united to the jejunum by the Allen technic. The proximal portion of the jejunum was anastomosed to the distal segment as a Roux Y procedure, thus reestablishing the continuity of the intestinal tract. Two years after this operation another intervention became necessary, after which the patient made excellent progress for about six weeks. Later it became evident that more drastic measures would be necessary in order to provide a better outlet for the flow of bile, which had been obstructed for so long by recurrent strictures at the hilus of the liver. With the experimental and surgical evidence of an interlobar communication of the ducts reported by Longmire as a background, it was decided to remove the entire left lobe of the liver, isolate the left hepatic duct, determine its communication with the right lobe and anastomose the end of the left duct to the side of the jejunum. This was done. The convalescence was complicated by bile peritonitis, but this cleared. After this the condition of the patient was better than it had been at any time since the cholecystectomy. The demonstration of an interlobar communication of the ducts in this case seems to substantiate the views expressed by Longmire. Technically, the operation described is most difficult and the risk is great. Fortunately, the indications for surgical measures of such magnitude are rare.

Arizona Medicine, Phoenix

6:1-80 (May) 1949

Critical Analysis of Aseptic Technic for Tuberculosis. W. H. Oatway Jr. —p. 23.
Q Fever. A. G. Bower.—p. 30.
Cardiovascular Epistaxis. E. H. Brown.—p. 33.
A.M.A.'s Plan of Battle: Outline of Strategy and Policies in Campaign Against Compulsory Health Insurance. C. Whitaker and L. Baxter. —p. 41.
Why I Must Participate in Socialized Medicine—If It Comes. F. N. Darby.—p. 45.
Control of Tuberculosis from World Viewpoint. J. E. Perkins.—p. 47.

6:1-80 (June) 1949

Presidential Address. R. S. Finn.—p. 15.
Infectious Mononucleosis. A. G. Bower.—p. 17.
Bundle Branch Block with Spontaneous Remission After Eighteen Months. W. H. Bates.—p. 21.
Review of Thromboangiitis Obliterans. L. Manoil.—p. 24.

Bulletin of Johns Hopkins Hospital, Baltimore

84:497-606 (June) 1949

Factor in Old Hepatitis Serum Capable of Agglutinating Chicken Red Cells. F. B. Bang.—p. 497.
Influence of Sex upon Lethal Effects of Hepatotoxic Alkaloid, Monocrotaline. O. D. Ratnoff and G. S. Mirick.—p. 507.
Attempted Heterologous Transplantation of Carcinoma of Cervix. R. B. Scott and C. B. Brack.—p. 526.
Observations on Effects of Tetraethyl Pyrophosphate (TEPP) in Man, and on Its Use in Treatment of Myasthenia Gravis. D. Grob and A. McG. Harvey.—p. 532.
Histochemical Studies on Nucleic Acid Phosphatase. J. S. Friedenwald and J. E. Crowell.—p. 568.

Canadian J. of Research. Medical Sciences, Ottawa

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Agglutination of Erythrocytes Containing Sulfhemoglobin—Further Evidence of Specific Aggregation. K. W. McKerns and O. F. Denstedt. —p. 164.
Effect of Antitoxin on Experimental Infection with Clostridium Novyi (Oedematiens) in Guinea Pigs, with Note on Active Immunization. G. G. Waters and P. J. Moloney.—p. 171.
Studies on Effect of Some Aromatic Amidines and Other Chemical Compounds on Growth of Influenza Virus in Embryonated Egg. L. McClelland and C. E. van Rooyen.—p. 177.
Relationship of Serum Phosphatases to Sex Hormones. J. Tuba, D. B. Baker and M. M. Cantor.—p. 202.

Diseases of Chest, Chicago

16:1-128 (July) 1949. **Partial Index**

*Use of Para-Aminosalicylic Acid in Chronic Pulmonary Tuberculosis. C. Eastlake Jr. and A. L. Barach.—p. 1.
Streptomycin in Tuberculosis Clinical Experience. M. Albertal.—p. 15.
Extrapleural Pneumonolysis with Plombage Versus Thoracoplasty. H. E. Walkup and J. D. Murphy.—p. 18.
Pneumoperitoneum Therapy in Lower Zone Tuberculosis. F. W. Lyons. —p. 21.
Carcinoma of Lung with Nonmalignant Pleural Effusion: Recovery by Pneumonectomy. A. Goldman.—p. 29.
Nutrition Education in Institutions Caring for Tuberculous Patients. M. S. Follstad and J. B. Novak.—p. 33.
Sterile Hemopneumothorax Caused by Softening and Perforation of Pulmonary Infarct. J. M. Masson and S. A. Hartman.—p. 42.
*Spontaneous Perforation of Normal Esophagus. P. F. Ware and J. W. Strieder—p. 49.
Chronic Constrictive Tuberculous Pericarditis: Report of Case with Pericardiectomy. F. A. Hughes and S. Lipton.—p. 66.
Intrathoracic Sympathoblastoma. M. R. Louria.—p. 75.
*Bronchial Lavage in Tuberculosis. J. J. Castillo.—p. 81.
Cerebral Air Embolism Associated with Spontaneous Pneumothorax. I. Kass and S. H. Dressler.—p. 86.
Dusts of Clinical Significance. T. M. Frank.—p. 89.
Use of Bronchoscopy in Bronchiectasis. F. D. Woodward and M. L. White Jr.—p. 94.
Tomography of Larynx in Disseminated Pulmonary Tuberculosis. M. Espinoza Galarza.—p. 100.

Para-Aminosalicylic Acid in Chronic Pulmonary Tuberculosis.—Eastlake and Barach found that the serum of tuberculous patients and healthy subjects produces a definite inhibition of the growth of the tubercle bacillus in Dubos mediums after ingestion of 2 to 4 Gm. of para-aminosalicylic acid. This has been demonstrated with the H37Rv strain as well as with strains recovered from patients with active pulmonary tuberculosis. Para-aminosalicylic acid was administered to 12 patients with advanced chronic pulmonary tuberculosis. The dosage employed was 10 to 11 Gm. daily in courses of three weeks with a free interval of one week. Observable clinical improvement took place in all 7 patients in whom the symptoms of active infection were sufficiently pronounced to permit judgment of the effectiveness of the drug. In 3 patients there was recurrence of symptoms on cessation of treatment. Three patients who had fever and signs of acute exudative tuberculosis were much improved and in good general condition after treatment, but their disease was not arrested. The characteristic effects of administration of para-aminosalicylic acid were prompt reduction in cough and expectoration and decrease in fever. These favorable results were generally manifested within the first week of treatment. The side effects of the drug were intermittent nausea, vomiting and looseness of the bowels. In 3 of the 12 patients these symptoms were sufficiently severe to force discontinuance of the drug. The favorable clinical results of treatment with para-aminosalicylic acid in chronic pulmonary tuberculosis suggest that this drug is a useful agent in the treatment of acute exacerbation of the disease.

Spontaneous Perforation of Normal Esophagus.—Ware and Strieder emphasize that spontaneous perforation is a distinct clinical entity in which sudden rupture occurs through all layers. The laceration occurs usually in a middle-aged, alcoholic man and after violent retching or vomiting. Acid gastric contents and food particles are driven into the mediastinum, left pleural cavity and occasionally also into the right pleural cavity. The features of the disease are distinctive, yet the diagnosis has rarely been made during life. The mortality has been 100 per cent until recently. The laceration classically occurs in the lower fifth of the esophagus, in a longitudinal direction and on the left posterolateral aspect; it is sharp edged and incised in appearance. Mediastinitis, empyema and pneumothorax usually result immediately. Roentgenograms of the chest, thoracentesis and subcutaneous emphysema enable one to make a correct diagnosis. The authors present the case of a man, aged 53, in whom a posterior mediastinotomy was done as a desperate measure when the patient was dying. The patient died. The authors outline a rationale of treatment which involves supporting care with early thoracotomy and closure of the laceration by suture.

Bronchial Lavage in Tuberculosis.—Tuberculous surveys made by Castillo since 1940 proved the inadequacy of roentgenologic examination alone to prove or disprove a tuberculous origin of pulmonary disease in patients with no expectoration.

He obtained secretions directly from the bronchi but gave up the method because it proved unsatisfactory. Examination of the contents of the fasting stomach likewise proved unreliable. Since 1943 he used bronchial lavage, employing the technic he had developed for bronchographies. From 1945 to 1948 a total of 2,414 bronchial lavages were performed in Cuba, of which 614 were done by Castillo. He found that in 45.4 per cent of cases of moderately advanced disease considered as cured, tubercle bacilli were still found by means of bronchial lavage. Roentgenograms in these cases showed evidence of fibrosis only. Bronchial lavage revealed tubercle bacilli also in 17 per cent of patients with moderately advanced disease who had been discharged as cured with normal roentgenograms. Patients with minimal lesions with no expectoration gave positive results with bronchial lavage in 90 per cent of the cases. In 10 per cent of contact cases with no roentgenographic evidence of tuberculosis, bronchial lavage showed tubercle bacilli to be present. Tubercle bacilli were not demonstrated by bronchial lavage in 90 cases of bronchial asthma, pulmonary cancer and bronchiectasis.

Endocrinology, Springfield, Ill.

44:487-600 (June) 1949. **Partial Index**

Modified Pettenkofer Reaction for Quantitative Estimation of Dehydroisoandrosterone and Its Application to Analysis of Urinary Extracts and Fractionations. L. Hansen.—p. 492.
Relative Growth of Thyroid Gland in Bovine Fetus. C. W. Nichols Jr., I. L. Chaikoff and J. Wolff.—p. 502.
Relationships of Diet to Duration of Survival, Body Weight and Composition of Hypophysectomized Rats. J. H. Shaw and R. O. Greep. —p. 520.
Mode of Action of Estrogens on Corpus Luteum. E. O. Höhn and J. M. Robson.—p. 536.
Quantitative Bio-Assay of Chorionic Gonadotrophin with Male Frog. A. L. Haskins Jr. and A. I. Sherman.—p. 542.
Comparative Activity of Thiouracil and Other Antithyroid Compounds in Rhesus Monkey. D. A. McGinty and M. L. Wilson.—p. 546.
Effect of Adrenal Cortex Extract With and Without Epinephrine upon Work of Adrenally Insufficient Rats. D. J. Ingle and J. E. Nezamis. —p. 559.
New Reagent for Histochemical Demonstration of Active Carbonyl Groups. New Method for Staining Ketonic Steroids. R. Ashbel and A. M. Seligman.—p. 565.
New Reagent for Histochemical Demonstration of Active Carbonyl Groups. Preparation of 2-Hydroxynaphthalene Carboxylic and Sulfonic Acid Hydrazides. A. M. Seligman, O. M. Friedman and J. E. Herz.—p. 584.

Hawaii Medical Journal, Honolulu

8:323-396 (May-June) 1949

Racial Incidence of Mental Disease in Hawaii. B. Wedge and S. Abe. —p. 337.
Keratoconjunctivitis Due to Crownflower. W. W. Wong.—p. 339.
Cytologic Diagnosis of Cancer by Smear Technic: II. Technical Methods. I. L. Tilden.—p. 342.
Multiple Transfusions in Chronic Lymphatic Leukemia. L. Welty and E. K. Maeda.—p. 246.
Paratyphoid A Fever: First Proven Case in Hawaii. W. O. French and P. Kim.—p. 350.

International Journal of Leprosy, New Orleans

16:421-532 (Oct.-Dec.) 1948. **Partial Index**

*"Spotted" Leprosy of Lucio: Introduction to Its Clinical and Histological Study. F. Latapí and A. Chevez Zamora.—p. 421.
*Tuberculosis and Leprosy, Antagonistic Diseases: Eviction of Leprosy by Tuberculosis. R. Chaussinand.—p. 431.
Intradermal Administration of Promin. G. Herrera.—p. 439.
Studies on Reticulum in Different Types of Leprosy. M. Vegas, J. Convit, and J. Espin.—p. 443.
Comparative Study of Nerve Branches of Skin in Tuberculoid and Lepromatous Leprosy. A. Castañé Decoud.—p. 451.

Spotted Leprosy of Lucio.—Latapí and Chevez Zamora point out that the existence in Mexico of a special clinical form of leprosy was described by Lucio in 1852 under the name of "manchada" or "lazarine" leprosy. For more than half a century this form of leprosy was forgotten, but it was verified in 1938 by Latapí. He reintroduced the term calling it "a pure and primitive diffuse lepromatosis with outbreaks of multiple necrotizing vasculitis." Modern Mexican leprologists have confirmed Lucio's description, particularly as to the "red and painful spots undergoing necrosis," and have completed its description by calling attention to diffuse infiltration of the skin, telangiectasis, cephalic alopecia, bacteriologic observations, false positive serologic reactions for syphilis, lepromatous structure, vascular location of the acute lesion and a special reaction to lepromin. They have observed its remarkable control and spectacular improvement by sulfone treatment. The authors summarize the essential facts pertaining to this form of leprosy and present the work being done in Mexico, particularly that which refers to the comparative clinical and histopathologic study of (*a*) the diffuse cutaneous infiltration, which they have found to be different from the classic nodular type, and (*b*) the acute episodic lesions, which are essentially dependent on an acute vascularitis and for which Latapí has proposed the name "Lucio's phenomenon" or "erythema necrotisans." The authors describe the Medina reaction, a special response to lepromin exhibited by patients with Lucio's phenomenon.

Tuberculosis and Leprosy, Antagonistic Diseases.—Chaussinand comments on the disappearance of leprosy from central and western Europe after the crusades and after the pandemics of pestilence in the fourteenth century. Numerous theories have been advanced to explain the disappearance of leprosy. The study of allergy in tuberculosis and leprosy, carried out during the last decade, reveals the cause for the decline in leprosy. Tuberculosis and leprosy are antagonistic diseases: one can observe the existence of relative cross immunity proper to these two infections. Tuberculosis is much more contagious and virulent than is leprosy in human beings. The tubercle bacillus spreads more rapidly and decisively in all regions in which the organisms of tuberculosis and leprosy enter into competition. Since a first attack by the tubercle bacillus provides a certain degree of antileprous immunity, persons infected with or who have had contact with Mycobacterium tuberculosis find themselves in a state of relative protection against a later attack by Mycobacterium leprae. The progressive eviction of leprosy by tuberculosis is the result.

Jour. of Industrial Hygiene & Toxicology, Baltimore

31:123-174 (May) 1949

*Acute Pneumonitis in Workers Exposed to Beryllium Oxide and Beryllium Metal. J. C. Aub and R. S. Grier.—p. 123.
Absorption and Excretion of Fluorides. III. Further Observations on Metabolism of Fluorides at High Levels of Intake. E. J. Largent and F. F. Heyroth.—p. 134.
Benign Pneumoconiosis in Tin Oxide Recovery Plant. H. C. Cutter, W. W. Faller, J. B. Stocklen and W. L. Wilson.—p. 139.
Physiologic Response of Animals to Respiratory Exposure to Vapors of Diisopropylamine. J. F. Treon, H. Sigmon, K. V. Kitzmiller and F. F. Heyroth.—p. 142.
Local and Systemic Effects Following Skin Contact with Phenol—Review of Literature. W. B. Deichmann.—p. 146.
Observations on Percutaneous Absorption of Toxaphene in Rabbit and Dog. II. R. W. Lackey.—p. 155.
Toxicity of Thioacetamide in Rats. A. M. Ambrose, F. DeEds and L. J. Rather.—p. 158.
Hazards Associated with Use of Lead Arsenate in Apple Orchards. L. M. Farner, C. D. Yaffe, N. Scott and F. E. Adley.—p. 162.

Acute Pneumonitis in Workers Exposed to Beryllium.—Aub and Grier saw in consultation 7 patients with acute pneumonitis from a laboratory where only two forms of beryllium were used, beryllium oxide and metallic beryllium. Both these substances were in a high degree of purity. Exposure to fumes of fluoride, sulfate or chloride was not a factor. These cases support the view that beryllium ion is toxic in its own right. It is not correct to attribute the disease associated with soluble beryllium salts to the acid radicals.

Journal of Investigative Dermatology, Baltimore

12:317-354 (June) 1949

Effective Approach in Treatment of Acne. K. A. Baird.—p. 317.
Reducing Substance in Urine of Patients Receiving Sodium Para-Aminobenzoate. R. H. Grekin and C. J. D. Zarafonetis.—p. 319.
Inhibition of "Itchy Skin" with Tetraethylammonium. R. R. Sonnenschein, T. Cornbleet, H. Janowitz and M. I. Grossman.—p. 321.
Quality, Intensity and Field Distribution of Radiation Emitted from New Type of Low Voltage Roentgen Therapy Tube. H. M. Lewis and A. Mutscheller.—p. 325.
Subcutaneous Fat Necrosis of the Newborn: Certain Etiologic Considerations. R. O. Noojin, B. F. Pace and H. G. Davis.—p. 331.
Combined Effect of Sulfanilamide and Penicillin in Treatment of Experimental Erysipelothrix Rhusiopathiae Infection of Mice. J. V. Klauder and A. M. Rule.—p. 335.
Local Injections of Preparation of Hyaluronidase in Treatment of Mucinous Infiltration of Skin. D. Bloom, F. J. Herrmann and H. Sharlit.—p. 339.
Local Injections of Preparation of Hyaluronidase in Treatment of Localized (Pretibial) Myxedema. M. L. Grais.—p. 345.

Journal of Nervous and Mental Diseases, New York

109:383-474 (May) 1949

Electroencephalogram After Head Injury. I. C. Kaufman and A. E. Walker.—p. 383.
Varieties of Electric Shock Therapy. B. L. Pacella.—p. 396.
Suppression of Motor Phenomena of Electroshock and Electronarcosis by Modification of Current Level. Esther Bogen Tietz, C. W. Olsen and W. R. Rosanoff.—p. 405.
Heredofamilial Cerebellar Ataxia with Spastic Paralysis and Spinal Muscular Atrophy: Report of Two Cases of Unusual Syndrome. E. D. Kilbourne.—p. 409.
Auditory Hallucinations and Subvocal Speech: Objective Study in Case of Schizophrenia. L. N. Gould.—p. 418.
Infusion of Fluids into Subarachnoid Space of Human Beings. L. Linn. —p. 428.
Diagnosis and Rational Psychotherapy. C. H. Patterson.—p. 440.
Management of Neurosyphilis. C. E. Johnson Jr.—p. 451.

109:475-570 (June) 1949

Combat Exhaustion: Descriptive and Statistical Analysis of Causes, Symptoms and Signs. R. L. Swank.—p. 475.
Statistical Analysis of 480 Army Psychiatric Casualties. J. J. Geller. —p. 509.
Psychobiologic Dysfunction in Mental Disease. J. R. Jacobson.—p. 519.
*Electroshock and other Therapeutic Considerations in Sexual Psychopathy. G. N. Thompson.—p. 531.
Unidirectional Electro-Stimulated Convulsive Therapy: II. Therapeutic Results in 536 Patients. E. Friedman.—p. 540.
Further Contribution to Psychodynamics of Convulsive Treatment. J. Flescher.—p. 550.

Electroshock in Sexual Psychopathy.—From the histories of 6 patients Thompson concludes that convulsive shock therapy is of value in the treatment of psychosis with psychopathic personality but that it does not alter the basic characteristics of the psychopathic personality. Sexual psychopathy is not altered by convulsive shock therapy. The symptoms of the psychopathic personality are not due to newly formed engrams as are the symptoms of the functional psychoses; otherwise convulsive shock therapy would be expected to prove of some value. The symptoms of the psychopathic personality are due to malfunctioning engrams or to a release mechanism by which unknown centers are allowed to discharge without normal cortical control.

Journal of Urology, Baltimore

61:981-1124 (June) 1949. Partial Index

Mixed Hypernephroid Tumors of Kidney (Metanephromas). C. G. Tedeschi, W. H. Holtham and C. L. Minor.—p. 981.
Associated Kidney and Bladder Tumors. A. F. Kaminsky.—p. 997.
*Use of Pyrogens in Treatment of Lower Nephron Nephrosis. L. J. Scheinman.—p. 1003.
"Trueta" Renal Vascular "Shunt": Experimental Demonstration of Neurovascular Control of Renal Circulation in the Rabbit, Cat, Dog and Monkey. W. E. Goodwin, R. D. Sloan and W. W. Scott.—p. 1010.
Roentgenographic Observation of Perforation of Kidney Following Retrograde Pyelography. A. Druckmann and S. Schorr.—p. 1028.
Easily Sterilizable, Flexible Cassette with Intensifying Screens, Designed for X-Ray Examination of Operatively Exposed Kidneys. O. Olsson. —p. 1032.
Contribution to Differential Diagnosis of Neoplasm of Bladder. S. Wesolowski.—p. 1034.
Analysis of Regional Variations in Response of Detrusor Muscle to Electrical Stimulation of Hypogastric Nerves. E. S. Hegre and E. H. Ingersoll.—p. 1037.
Enuresis: Report of 2 Interesting Cases. W. M. Flagg.—p. 1048.
Treatment of Hypospadias. F. Z. Havens and A. S. Black.—p. 1053.
Chorio-Epithelioma of Testicle with Report of Case. R. L. Atkinson. —p. 1068.
Bilateral Lympho-Epithelioma of the Testicle and Epididymis, Metastatic from the Nasopharynx. G. K. Kawaichi.—p. 1073.
*Post-Mortem Compared with Clinical Diagnosis of Genito-Urinary Tuberculosis in Adult Males. E. M. Medlar, D. M. Spain and R. W. Holliday.—p. 1078.
Müllerian Duct Cysts. R. R. Landes and C. L. Ransom.—p. 1089.
Number of Motile Spermatozoa as Index of Fertility in Man: Study of 406 Semen Specimens. E. J. Farris.—p. 1099.
Sulfadiazine-Sulfacetimide Mixture in Treatment of Urinary Tract Infections. H. Seneca, E. Henderson and M. Harvey.—p. 1105.

Pyrogens in Treatment of Lower Nephron Nephrosis.—Scheinman regards it as physiologically sound to attempt to increase the renal blood flow before irreversible ischemic necrosis of the tubules is widespread. He reviews the present concept of renal shutdown with emphasis on the theory that diminished renal blood flow is the initiating and chief perpetuating factor in this syndrome. He attempted to reverse the syndrome of renal shutdown by increasing renal blood flow with pyrogens. The studies were made on rabbits, of which 8 responded with oliguria to the intravenous injection of 65 cc. of sterile water. All 8 animals had transient hematuria. In the controls the oliguria persisted for at least three days. In 4 rabbits in which treatment with typhoid vaccine was started eighteen hours after the onset and repeated at twelve hour intervals, normal urinary output was resumed on the second day. The rabbit may not be the most desirable experimental animal for this problem because of its efficient reticuloendothelial system.

Diagnosis of Urogenital Tuberculosis.—Medlar and his associates believed that routine necropsy records would provide data on the pathogenesis of tuberculosis. The routine necropsy protocoles at Bellevue Hospital are valuable because there is a Chest Service that serves as a clearinghouse for tuberculous patients. An analysis of 5,424 necropsies on males over 16 years of age revealed that tuberculous lesions in the genitourinary system were present in 3.1 per cent of all necropsy material, in 4.5 per cent of the persons who harbored unhealed tuberculous lesions but died from causes other than tuberculosis and in 26 per cent of persons dead from tuberculosis. Caseous or cavitating pulmonary tuberculous lesions were present in 85 per cent of those with genitourinary lesions. Forty-five per cent of the persons with tuberculous lesions in the genitourinary organs died from generalized miliary tuberculosis. Lesions in the kidney were twice as frequent as those in the prostate and three times as frequent as those in the seminal vesicle or epididymis. The incidence of genitourinary lesions was greater in Negro than in white males. Sixty-five per cent of the renal lesions were miliary in type, with 98 per cent bilateral; in this group 13 per cent showed lesions in the genital system. Twenty-three per cent of the renal lesions were caseous in type, with 86 per cent bilateral; in this caseous group 52 per cent revealed lesions in the genital system. Twelve per cent of the renal lesions were cavitating in type, with bilateral lesions in 58 per cent; in this cavitating group lesions in the genital system were present in every case. In tuberculosis of the genital system the prostate, seminal vesicle and epididymis were all involved in 63 per cent, the prostate alone in 29 per cent and the seminal vesicle or epididymis alone in no case. The clinical recognition of tuberculous disease in the genitourinary organs occurred in only 18 per cent. This indicates the difficulty in recognizing it. Advanced tuberculous disease may be present without significant urologic symptoms.

Michigan State Medical Society Journal, Lansing

48:649-792 (June) 1949

Psychotherapy Reviewed. E. J. Alexander.—p. 697.
Rise in Hospital Costs. E. D. Barnett.—p. 707.
Narcotherapy in Psychosomatic Disorders. G. S. Evseeff.—p. 709.
The Climacteric and Its Management. H. H. Cummings. —p. 713.
Acute Bacterial Endocarditis Complicating Pregnancy. G. N. Ferris and C. E. Toshach.—p. 717.
Random Relationships of Experimental Embryology and Genetics to Pathology. S. P. Reimann.—p. 720.
New Drugs for Allergic Diseases. G. L. Waldbott and J. J. Gadbaw. —p. 724.
Familial Repetition of Myelomeningocele. I. A. Wilner.—p. 727.
Antipertussis Rabbit Serum in 95 Cases and 15 Contacts: Preliminary Report. A. R. Ceresko and H. A. Raskin.—p. 728.

Nebraska State Medical Journal, Lincoln

34:201-236 (June) 1949

Embodiment of an Idea. J. E. M. Thomson.—p. 204.
Individualizing Transfusion. B. C. Russum and D. D. McLean.—p. 208.
Fluorescent Light Compared with Incandescent Light. H. O. Paulson. —p. 212.
Beryllium Poisoning: Its Increasing Clinical Importance and Report of Case of Pulmonary Granulomatosis Due to Beryllium. F. L. Dunn and P. T. Pratt.—p. 217.

34:237-276 (July) 1949

Surgical Principles in Care of Severely Burned. C. W. McLaughlin Jr. and R. M. Cochran.—p. 240.

SYMPOSIUM ON GYNECOLOGIC PROBLEMS

Sterility. R. Luikart.—p. 245.
Uterine Displacements. E. C. Sage.—p. 247.
Functional Uterine Bleeding. J. J. Freymann.—p. 250.
Ectopic Pregnancy. H. E. Anderson.—p. 254.
Common Benign Lesions of Cervix. J. J. Grier.—p. 256.

New England Journal of Medicine, Boston

240:947-986 (June 16) 1949

*Auricular Fibrillation in Normal Hearts. H. H. Hanson and D. I. Rutledge.—p. 947.
*Schick Status of 18,000 Young Adult Males. J. Worcester and F. S. Cheever.—p. 954.
Sequelae of Eastern Equine Encephalomyelitis. J. C. Ayres and R. F. Feemster.—p. 960.
Hepatitis, Exfoliative Dermatitis and Abnormal Bone Marrow Occurring During Tridione Therapy. S. E. Leard, W. E. R. Greer and I. C. Kaufman.—p. 962.
Public-Health Aspects of Virus Encephalitides. J. C. Ayres and R. F. Feemster.—p. 966.
Regional Ileitis. Hepatic-Vein and Vena-Cava Thrombosis (Chiari's Syndrome).—p. 975.
Rheumatic Heart Disease, Aortic Stenosis and Regurgitation. Congenital Anomaly—Single Pelvic Kidney. Chronic Glomerulonephritis.—p. 979.

Auricular Fibrillation in Normal Hearts.—Hanson and Rutledge reviewed a series of 651 cases of auricular fibrillation seen at the Lahey Clinic and found that only 30 cases (4.6 per cent) occurred in normal hearts. That auricular fibrillation occurs in normal hearts has been proved repeatedly at postmortem examination. Yater emphasized that there is no pathologic change in the heart characteristic of auricular fibrillation. Other investigators demonstrated that in normal or controlled auricular fibrillation the cardiac output and the rate of blood flow are not diminished and the blood pressure is constant, insuring adequate tissue oxygenation. Of the 30 patients, 20 have been adequately followed and 13 of these still show no evidence of heart disease. In only 7 has evidence of heart disease developed, and in these the manifestations occurred only after years of asymptomatic fibrillation and as the subjects were approaching senility. These patients should receive an adequate explanation of their condition and should be reassured that it is benign. If precipitating factors can be determined in the paroxysmal type of fibrillation, an attempt should be made to remove them and the patient should be advised to get more rest, relaxation and diversion of interest. The usual case of paroxysmal auricular fibrillation in which the patient has infrequent attacks of short duration is not benefited by therapy. The author comments on the use of quinidine and digitalis and says that in more than a third of the patients reported here no treatment was given. Digitalis had no effect in a fifth of the cases in which it was used, and quinidine was ineffective in a fourth of the cases. The prognosis depends on the condition of the myocardium; the arrhythmia itself appears to be without significance. Treatment is not indicated unless complications develop, and the therapy is that of the complication.

Schick Status of Young Adults.—The recent renewed increase in diphtheria together with the shift in age distribution induced Worcester and Cheever to present the results of a Schick survey among some 18,000 naval recruits carried out during the winter of 1941 and 1942. The recruits received their Schick tests promptly after their induction into naval service. Consequently, the results of the tests reflect civilian rather than military experience with diphtheria. The Schick test was carried out by injection intradermally into one forearm of 0.1 cc. of a standard diphtheria toxin so diluted that 0.1 cc. contained one-fiftieth MLD. A Moloney test was performed at the same time by the injection of 0.1 cc. of a 1:100 dilution of diphtheria toxoid into the other forearm by the intradermal route. The tests were read at the end of forty-eight hours and again at the end of one hundred and twenty hours. There are differences in the incidence of positive reactors in the different parts of the country, but there is no close correlation between the percentages of the population susceptible to diphtheria in this young adult group and the morbidity rates for the total population. The New England and Middle Atlantic regions combine low morbidity with high susceptibility, whereas the South shows a low susceptibility combined with a high morbidity. In the West, both the morbidity rates and the susceptibility seem to be high. These statements should not be interpreted in terms of the natural history of the disease without knowledge of the immunization programs in the various sections. The results of surveys on immunization are not entirely reliable. The evidence suggests that whatever role active immunization may have played in reducing diphtheria morbidity, it has had little effect on the immunity status of the general population. At least in some regions of the country the present Schick status appears to be directly related to the amount of clinical disease experienced in the last ten years. The authors conclude that the low level of immunity as evidenced by the high percentage of Schick-positive reactors among young men is not a cause for complacency.

New Jersey Medical Society Journal, Trenton

46:269-312 (June) 1949

Diagnosis and Management of Small Bowel Obstruction. E. V. Parsonnet.—p. 270.
Contact Dermatitis Due to Topical Anesthetics. M. Braitman.—p. 274.
Rheumatism. R. E. Humphries.—p. 277.
Neonatal Hazards. R. A. Weinstein.—p. 279.
Annual Examination for Your Patient. R. B. Marin.—p. 285.
Role of X-Ray Therapy in Non-Tumorous Conditions. M. P. Mains.—p. 287.
Solitary Eosinophilic Granuloma of Bone. R. R. Goldenberg and V. W. Guidice.—p. 292.
Clostridium Histolyticum Infection. W. H. Buffey, C. R. Riley and J. R. Massie Jr.—p. 294.
Pregnancy Complicated by Lymphogranuloma Venereum with Colostomy. J. M. Smolev and A. B. Tamis.—p. 297.

New York State Journal of Medicine, New York

49:1361-1486 (June 15) 1949

Hypertensive Encephalopathy and Cerebral Arteriosclerosis. H. I. Russek and B. L. Zohman.—p. 1411.
Amputation in Patients with Diabetes Mellitus. J. S. West and E. M. Pepper.—p. 1415.
Acute Coronary Occlusion Simulating Acute Abdominal Disease. L. E. Field, L. Pordy and A. M. Master.—p. 1419.
Cerebral Complications Resulting from Hypertension Caused by Vasopressor Drugs in Obstetrics. B. A. Greene and J. Barcham.—p. 1424.
Practical Aspects of Control of Dicumarol Therapy. L. E. Blumenthal.—p. 1427.
White and Nonwhite Maternal Mortality in Brooklyn, New York, 1947. C. A. Gordon.—p. 1431.
Salt-Poor Diet. M. Plotz.—p. 1439.
Modification of the Slide Rh Determination. I. B. Cohen and H. Pretshold.—p. 1441.
Pulmonary Coccidioidomycosis. F. W. Holcomb.—p. 1443.
Incidence of Multiple Primary Malignant Tumors and Report of Case with Three Primary Carcinomata. D. S. Spain.—p. 1445.

Proc. of Staff Meet. of Mayo Clinic, Rochester, Minn.

24:341-364 (June 22) 1949

*Increase in Resistance of Tubercle Bacilli to Sodium *P*-Aminosalicylic Acid: Observations on Cultures Isolated from Patients During Chemotherapy. A. Delaude, A. G. Karlson, D. T. Carr and others.—p. 341.
Polythene Prosthesis to Replace Lung After Pneumonectomy. J. H. Grindlay, O. T. Clagett and A. H. Bulbulian.—p. 346.
Cardiac Clinics. CXXVIII. Origin and Evolution of Diagnostic Procedures with Reference to Diseases of Heart and Circulation: I. Pulse. F. A. Willius.—p. 350.
Cough and Asthma. L. E. Pickman.—p. 354.
Management of Pregnancy Complicated by Toxic Goiter: Report of Case. W. H. Jondahl, E. A. Banner and L. P. Howell.—p. 358.

Resistance of Tubercle Bacilli to Sodium *P*-Aminosalicylic Acid.—Delaude and his associates of the Mayo Clinic point out that one of the problems created by the increasing use of para-aminosalicylic acid (PAS) in the treatment of clinical tuberculosis is the question of the possible occurrence of tubercle bacilli resistant to this drug. In vitro tests were done with sodium *p*-aminosalicylic acid (NaPAS) because of the low solubility of para-aminosalicylic acid. Cultures from 71 patients who had not received para-aminosalicylic acid treatment were resistant to only 0.006 to 0.012 mg. of sodium *p*-aminosalicylic acid per hundred cubic centimeters of egg yoke agar medium. Cultures from 18 patients treated with para-aminosalicylic acid alone for 94 days or less were likewise resistant to 0.006 to 0.012 mg. of sodium *p*-aminosalicylic acid per hundred cubic centimeters of medium both at the beginning and at the end of therapy. Cultures isolated from 4 of 5 patients treated with para-aminosalicylic acid for periods of 157 to 251 days were able to grow in concentrations of sodium *p*-aminosalicylic acid of 1.6 to 6.4 mg. per hundred cubic centimeters of medium. Cultures from 4 patients who received approximately the same amount of para-aminosalicylic acid in combination with promin® (sodium p,-p'-diaminodiphenylsulfone-N, N'didextrose sulfonate) and streptomycin did not show this

increase in resistance to sodium *p*-aminosalicylic acid in vitro. Tubercle bacilli of 2 of the patients first became resistant to streptomycin during streptomycin therapy and later became resistant to sodium *p*-aminosalicylic acid during treatment with para-aminosalicylic acid. The clinical significance of the occurrence of tubercle bacilli resistant to sodium *p*-aminosalicylic acid must be determined by in vivo studies in experimentally infected animals and in patients.

Psychoanalytic Quarterly, Albany, N. Y.

18:137-278 (April) 1949

Dynamics in Fetishism. M. E. Romm.—p. 137.
Dreams Following Intercourse. V. W. Eisenstein.—p. 154.
Origin of Clothes. A. Garma.—p. 173.
The Individual's Adjustment to Society. L. J. Saul.—p. 191.
Detective Stories and Primal Scene. G. Pederson-Krag.—p. 207.
Remarks on Comic Process and Beauty. S. Tarachow.—p. 215.
Fear of Mice. S. S. Feldman.—p. 227.

Public Health Reports, Washington, D. C.

64:749-784 (June 17) 1949

Professional Education for Cancer Control. A. V. Deibert.—p. 749.
National Cancer Institute Program of Postgraduate Training for Physicians. R. R. Spencer.—p. 750.
Cancer Teaching in Medical Schools. R. F. Kaiser.—p. 757.
Cancer Teaching in Dental Schools. R. F. Kaiser.—p. 764.

64:785-816 (June 24) 1949

*Rabies Problems and Control: Nation-Wide Program. J. H. Steele and E. S. Tierkel.—p. 785.
Pilot Mental Clinic: First Annual Report of Prince Georges County Clinic. Mabel Ross.—p. 797.

64:817-856 (July 1) 1949

Studies of Pulmonary Findings and Antigen Sensitivity Among Student Nurses: IV. Relationship of Pulmonary Calcification with Sensitivity to Tuberculin and to Histoplasmin. J. C. Goddard, L. B. Edwards and C. E. Palmer.—p. 820.
Characteristics of Commercial X-Ray Screens and Films—VII. W. W. Van Allen.—p. 847.

64:857-884 (July 8) 1949

Filariasis Control by DDT Residual House Spraying, Saint Croix, Virgin Islands: I. Operational Aspects. C. E. Kohler.—p. 857.
Id.: II. Results. H. W. Brown and R. W. Williams.—p. 863.
Salmonella Types Encountered in Maryland Between 1944 and 1948. A. A. Hajna.—p. 876.

Control of Rabies.—According to Steele and Tierkel, there has been an alarming increase in rabies in many parts of the country during the past fifteen years. Each year over 30,000 persons are required to take the long and often painful vaccine inoculations as a result of exposure to rabid or suspected dogs. The annual cost to the country for human vaccine treatments and livestock losses exceeds $5,000,000. The Subcommittee on Rabies, National Research Council, in 1945 and the National Conference on Rabies in 1947 agreed that nationwide uniformity of control procedures will be necessary for the eradication of the disease in the United States. The U. S. Public Health Service has offered its services to the states for a coordinated attack. The Rabies Control Branch, Veterinary Public Health Division, serves as the nerve center for rabies control. The authors discuss the laboratory diagnosis of the disease, canine rabies vaccination, reporting, the tasks of the state health departments and the local problem. It is stated that the local rabies control program includes three measures: impounding and destruction of all stray and ownerless dogs; annual antirabies vaccination of all dogs (the importance of canine vaccination is now firmly established and needs only an educational campaign to bring this fact to the public), and registration or licensing of all dogs. Except in heavily populated cities, vaccinated dogs, when properly tagged, may be allowed at large thirty days after vaccination. The vaccine is not fully effective until the end of that time. In an outbreak of rabies a strict quarantine should be placed on all dogs, requiring that they be confined at home or kept on a leash when out of doors. Dogs under 6 months of age are particularly susceptible to rabies and are not as readily immunized as adult dogs. They should be confined. Biting dogs and suspected rabid animals should be impounded and held under observation for twelve to fourteen days. Dogs exposed to rabies should be destroyed or kept confined under observation for six months. In case of rabies outbreaks in wild animals, such as foxes, adequate trapping programs should be instituted. Diagnostic facilities and human antirabies vaccine should be made available by the local health unit or branch laboratory of the state health department. All cases of rabies in human beings and animals should be reported to the local health officials.

Puerto Rico J. Pub. Health & Trop. Med., San Juan

24:293-406 (June) 1949

Quantitative Blood Cultures from Brucellosis Patients and from Animals Experimentally Infected with Brucella Organisms. C. Carrillo Cárdenas, H. Ordóñez and M. Ruiz Castañeda.—p. 293.
Action of Parasitic Amoeba and Antibiotic of Micrococcus on Brucella Cultures. O. Bonilla Soto and P. Morales Otero.—p. 305.
Heat-Stable, Water-Soluble Brucella Allergen: Preliminary Report. A. Pomales Lebrón.—p. 337.
Short Note on Epidemiology of Brucellosis in Puerto Rico. P. Morales Otero.—p. 349.
Treatment of Brucellosis with Sulfadiazine and Streptomycin. F. Hernández Morales.—p. 362.

Quarterly J. of Studies on Alcohol, New Haven, Conn.

10:1-184 (June) 1949

Administration of Alcoholism Rehabilitation Programs. S. D. Bacon.—p. 1.
Act of Surrender in Therapeutic Process with Special Reference to Alcoholism. H. M. Tiebout.—p. 48.
*Oxygen in Treatment of Acute Alcoholic Intoxication: Preliminary Clinical Report Based on Study of 100 Cases. C. N. Davis and H. F. Robertson.—p. 59.
Group Therapy in Alcoholism: Transcriptions of Series of Sessions Recorded in Outpatients Clinic: I. Introduction and First Two Sessions. R. G. McCarthy.—p. 63.
Patterns of State Taxation of Distilled Spirits with Special Reference to Kentucky. O. F. Traylor.—p. 109.
Joseph Turner and First Inebriate Asylum. S. Rypins.—p. 127.

Oxygen in Acute Alcoholic Intoxication.—During World War II Davis and Robertson observed military personnel who inhaled oxygen by mask for the relief of acute alcoholic intoxication. The results of this practice appeared sufficiently favorable to justify a study of the effects of pure oxygen inhalations on 100 patients. The subjects in this study were problem drinkers, including patients with hallucinosis and delirium tremens. The response was excellent. The treatment is applicable to acute alcoholic intoxication, but it does not alter the basic addiction to alcohol, nor can it prevent problem drinking. Immediately after admission, the patient is given pure oxygen by mask. The standard treatment consists of the administration of pure oxygen for twenty minutes every hour for six hours. Pure oxygen means a 63 per cent concentration of oxygen in the mask, as determined by chemical tests. For the occasional patient who has a severe psychotic episode and is suffering from delirium tremens the oxygen therapy is continued hourly for twenty-four to forty-eight hours until the patient has reacted favorably. No toxic reactions were noted. It is of vital importance that the personnel administering the oxygen be sympathetic and understanding and gain the confidence of the patient in order to avoid arousing fear by application of the mask. If the mask is applied without proper preparation, the patient may become alarmed and uncooperative and rebel against the treatment. The authors found sedatives necessary prior to the use of oxygen therapy. Since the institution of oxygen therapy they were able to treat 100 patients successfully without sedation. Critically ill patients will require additional supportive medication, such as the intravenous "Blockley cocktail" (providing dextrose, insulin, nicotinic acid and ascorbic acid) and measures to preserve adequate hepatic function. This study indicates a simple, practical therapeutic advance in the management of acute intoxication of the alcoholic person.

Review of Gastroenterology, New York

16:451-536 (June) 1949

Differential Diagnosis of Ulcerative Colitis. B. B. Crohn.—p. 463.
Medical Management of Ulcerative Colitis. M. Paulson.—p. 468.
Lysozyme Activity in Chronic Ulcerative Colitis. K. Meyer, A. Gellhorn, J. Prudden and others.—p. 476.
Surgical Therapy in Ulcerative Colitis. J. E. Rhoads.—p. 480.
Therapeutic Application of Anion Exchange Resins in Treatment of Peptic Ulcer. S. Weiss, R. B. Espinal and J. Weiss.—p. 501.

South Dakota Journal of Medicine, Sioux Falls

2:155-178 (May) 1949

Prolonged Labor: Its Management and Treatment. E. A. Banner.—p. 157.

2:179-214 (June) 1949

Role of Federal Government in National Cancer Program. R. L. Cherry.—p. 180.
The British Story—First Hand. E. Wilson.—p. 185.
Acute Surgical Conditions Complicating Pregnancy. E. G. Holmstrom.—p. 189.
Meckel's Diverticulum. J. V. McGreevy and E. J. McGreevy.—p. 195.

Southern Surgeon, Atlanta, Ga.

15:375-450 (June) 1949

Fundamental Principles of Thyroidectomy. M. Nordland.—p. 375.
Palliative Operations for Carcinoma of Pancreas. R. L. Sanders and C. H. Porter.—p. 383.
*Needle Biopsy of Liver in Diagnosis of "Surgical" Jaundice. J. P. Webb and S. Werthammer.—p. 393.
Subcapsular Nephrectomy—Its Indications and Advantages. W. E. Kittredge and M. Whitehead.—p. 402.
Placenta Accreta with Velamentous Insertion of Cord: Report of Case Treated by Hysterectomy Following Normal Delivery. R. E. Blount and L. U. Lumpkin.—p. 411.
*Congenital Absence of Pectoralis Major Muscle in Whole or in Part and Absence of Pectoralis Minor Muscle (Unilateral): Report of 3 Cases. W. M. Hayes.—p. 417.
Lead Poisoning in Infancy: Case in Which Two Operations were Performed on Stomach. W. L. Sibley.—p. 427.
Non-Obstructive Lesions of Colon. I. H. Lockwood and L. A. Gay.—p. 437.

Needle Biopsy of Liver.—Webb and Werthammer report 70 liver biopsies. At first, biopsies were done for the sake of standardization of histologic picture in a number of patients with liver enlargement or impaired hepatic function. More recently, the procedure has been employed only in patients with diagnostic problems with respect to the liver. The authors used the lateral intercostal approach with the "Vim"-Silverman needle. This approach is safer than the anterior approach. With the lateral approach the needle enters the liver in such a way as to avoid the hollow viscera and large abdominal vessels and even the larger hepatic veins. The authors observed a considerable hemorrhage in only 1 of their cases, and, although the patient died, the hemorrhage was not considered the cause of death. This patient had fulminant Weil's disease which was not diagnosed ante mortem. The authors discuss the microscopic aspects of liver biopsy and reproduce microphotographs of various pathologic processes. Diagnosis of extrahepatic obstruction can be made with certainty if biopsy is done within the first two or three weeks after the onset of jaundice. After this, in an occasional case there may be a histologic picture in the biopsy section which is extremely difficult to differentiate from long-standing hepatitis. In the majority of cases the clearcut picture of mechanical obstruction is still preserved so that the diagnosis of extrahepatic block may be confidently made even after several months of jaundice. The authors have performed 20 liver biopsies in 16 cases of jaundice in which reasonable doubt existed as to the mechanism of the jaundice. As shown by subsequent clinical course, surgical findings or necropsy, the correct diagnosis was made from the biopsy in 15 of the 16 cases.

Congenital Absence of Pectoralis Muscles.—Hayes reviews the literature on congenital absence of pectoralis muscles and describes 3 patients whom he had observed. The first patient was a man with congenital absence of the pectoralis major and minor muscles on the right side. The second patient was a man with congenital absence of the sternocostal portion of the pectoralis major muscle. The clavicular portion of the muscle was present and well developed. The pectoralis minor muscle was also absent on the right side. The third patient was a woman with congenital absence of the pectoralis major and minor muscles on the left side. There was little breast tissue, and this did not function. The woman also had bilateral cervical ribs. The sternocostal portion seems to be that portion which is most commonly missing.

Southwestern Medicine, El Paso, Texas

29:109-128 (June) 1949

Value of Post-Operative Radiation in Cancer of Breast.—When, Why and How. T. P. Eberhard and P. C. Swenson.—p. 112.
Mobile Lead Protective Screen with Film Storage Chest for Small X-Ray Room. R. D. Haire.—p. 118.

Urologic & Cutaneous Review, West Palm Beach, Fla.

53:321-384 (June) 1949. **Partial Index**

Neurogenic Bladder Associated with Herpes Zoster. W. A. Shullenberger and W. N. Wishard Jr.—p. 321.
Carcinoma of Lung Simulating Primary Carcinoma of Urinary Bladder: Case Report. J. S. Haines, H. Grabstald, V. A. McKusick and others.—p. 324.
Post-Caval Urether: Report of Case. J. W. Schwartz and N. S. Irey.—p. 327.
Renal Actinomycosis: Case Report. P. S. Rosenblum.—p. 329.
Dicumarol Therapy and Sequence of Gross Hematuria. W. A. Rosso and N. J. Heckel.—p. 332.
*Combination (Synergistic and Additive) Chemotherapy of Syphilis. J. A. Kolmer.—p. 338.
Common Skin Diseases of Summer Season and Their Management. M. Leider, E. H. Mandel, G. L. Popkin and C. Reiter.—p. 343.

Combination Chemotherapy of Syphilis.—Kolmer reviews the literature and his own observations on the synergistic and additive combined chemotherapy of syphilis in experimental animals and in human subjects. The following combined treatments have proved effective in acute syphilitic orchitis of rabbits: oxophenarsine hydrochloride plus a bismuth compound; penicillin plus oxophenarsine hydrochloride; penicillin plus a bismuth compound; penicillin plus oxophenarsine hydrochloride plus a bismuth compound, and penicillin plus iodides. Less effective results have been observed with tryparsamide plus penicillin and with tryparsamide plus a bismuth compound. In early syphilis of human beings the results of combination chemotherapy with penicillin plus oxophenarsine hydrochloride and with penicillin plus the arsenical plus a bismuth compound versus treatment with penicillin alone have been conflicting. Combination chemotherapy with penicillin plus oxophenarsine hydrochloride, penicillin plus a bismuth compound or with penicillin plus the other two agents may be preferable to penicillin alone in the treatment of primary and secondary syphilis and especially in the treatment of infectious relapses; likewise in the treatment of prenatal syphilis after the second trimester of pregnancy. From the standpoint of serious toxic reactions due to oxophenarsine hydrochloride, the treatment of early syphilis with penicillin plus this drug and with penicillin plus this drug plus a bismuth compound is safer than intensive treatment with the arsenical compound alone. Since it is doubtful that Treponema pallidum acquires resistance to the arsenical and bismuth compounds and penicillin in the treatment of syphilis, this theoretic objection to combination chemotherapy does not appear to be valid. The status of combination chemotherapy in the treatment of chronic experimental syphilis of rabbits and of late, latent, tertiary and congenital syphilis of human beings cannot be stated at the present time.

Virginia Medical Monthly, Richmond

76:327-380 (July) 1949

Vascular Hypertension. J. C. Hortenstine, W. P. McGuire, E. W. Lacy Jr. and M. D. Custer.—p. 329.
Diagnosis and Treatment of Carpal Injuries. G. Cooper Jr. and C. J. Frankel.—p. 336.
Injection Treatment of Internal Hemorrhoids. R. V. Terrell and C. C. Chewning Jr.—p. 340.
Acute Thrombocytopenic Purpura Following Massive Hormone Therapy in Pregnant Diabetic. D. B. Ridgway.—p. 344.
Influence of Stilbamidine in Multiple Myeloma with Pre-Existing Renal Involvement—Case Reports. J. D. Markham, J. R. Kriz and J. P. Williams.—p. 347.
Role of Liver in Blood Coagulation. M. L. Dreyfuss.—p. 356.
Surgical Treatment of Acute Gallbladder in First 72 Hours. M. Behrend.—p. 359.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

57:221-272 (June) 1949

Osteochondritis Dissecans of Head of Femur. F. L. Flashman and R. K. Ghormley.—p. 221.
Preoperative Scout Film of Chest. F. B. Wilkins, F. Isaac and R. E. Ottoman.—p. 229.
Arrhenoblastoma. K. A. O'Connor and A. W. Diddle.—p. 235.
Intravenous Therapy with Special Consideration of Parenteral Nutrition. C. O. Rice.—p. 238.
Tuberculosis of Endometrium Treated with Streptomycin: Report of Case. K. L. Schaupp Jr.—p. 243.
Measurement of Anterior Obstetrical Sagittal Diameter. J. C. McDermott.—p. 245.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted. Single case reports and trials of new drugs are usually omitted.

Archives of Disease in Childhood, London

24:81-158 (June) 1949

*Coeliac Disease: Relation Between Dietary Starch and Fat Absorption. W. Sheldon.—p. 81.
Blood Volume Studies in Healthy Children. S. J. M. Russell.—p. 88.
Some Etiological Factors in the Coeliac Syndrome. A. Brown.—p. 99.
Effect of Toxaemia of Pregnancy upon Foetus and Newborn Child. A. A. Brash.—p. 107.
The Nose in Relation to Cerebrospinal Fluid and Lymph Stream. J. M. Yoffey.—p. 117.
Renal Venous Thrombosis in the Newborn. M. L. Fallon.—p. 125.
Congenital Right-Sided Diaphragmatic Hernia: Some Difficulties in Differential Diagnosis and Operative Repair. R. Belsey and J. Apley.—p. 129.
Fugitive Pericarditis. O. D. Beresford.—p. 135.
*Congenital Toxoplasmosis: Report of 2 Cases. H. G. Farquhar and W. M. L. Turner.—p. 137.

Diet in Celiac Disease.—Sheldon stated in a previous communication reasons for regarding starch intolerance in children with celiac disease as of greater etiologic importance than the failure to absorb the products of fat digestion. Assuming that intolerance of starch exerts a primary etiologic role in celiac children and that failure to absorb fat is a secondary consequence dependent in some way on mismanagement of starch, the author decided that it might be possible to improve fat absorption by eliminating starch from the diet. He fed celiac children on two consecutive diets, both containing an ample portion of fat, the first diet containing starch and the second being free of starch. The absorption of fat was compared from each diet by means of fat balances. Fat absorption was much improved on a diet free from starch. The investigation was then reversed, beginning with a starch-free diet and later changing to one containing starch; again it was demonstrated that the inclusion of starch in the diet lowered the absorption of fat. Experiments on these lines have been conducted on 15 children with celiac disease. Withdrawal of starch from the diet was accompanied by a rise in the fat absorption that averaged 15 per cent. The starch-free diet enabled the children to tolerate virtually a normal intake and led to improvement in weight, in the character of the stools and in temperament. Within a few weeks the children were smiling, happy, playful and actively running about the ward. A four day period for a fat balance is too short when fat absorption is defective. Although a twelve day period was employed in this investigation, an eight day period would have been sufficient. Estimations of fecal fat alone do not provide a reliable index of fat absorption.

Congenital Toxoplasmosis.—According to Farquhar and Turner toxoplasmosis in man has been described in four forms, which vary with the age of the patient. The first is an infection affecting the fetus and the newborn child; this is the congenital type of the disease. The second form is recognized as an encephalitis affecting older children. The third form shows itself as an acute febrile exanthematic illness affecting adults. There is a mild and apparently symptomless type of infection, which may be recognized only by finding specific antibodies in the blood or by the patient's giving birth to an infected infant. The authors report 2 instances of congenital toxoplasmosis. Both patients showed the clinical manifestations of congenital toxoplasmosis. Serum antibody reactions were positive, but the organism has not been isolated. The finding of 2 such instances in one area within six months suggests that the condition may not be so rare in Britain as is at present believed. It would appear that the serologic investigation of patients showing some or all of the manifestations of this disease, in particular mental retardation, microcephaly and choroidoretinal degeneration in infancy, might well be extended. An investigation of a cross section of the population to determine the incidence of persons with positive reactions and their further examination would be of definite value.

British Medical Journal, London

2:41-112 (July 9) 1949

Sir William Osler Centenary. Some Recollections. A. S. MacNalty and others.—p. 41.
*Treatment of 160 Cases of Erythroblastosis Foetalis with Replacement Transfusion. J. J. van Loghem, J. H. van Bolhuis, J. M. Soeters and G. M. H. Veeneklaas.—p. 49.
Case of Anamnestic Reaction with Rh Agglutinins. D. A. Osborn.—p. 53.
*Carcinoma of Breast in Castrated Women. M. Dargent.—p. 54.
Congenital Hemiatrophy Associated with Other Congenital Defects. W. L. Calnan.—p. 56.

Replacement Transfusion in Erythroblastosis Fetalis.—Van Loghem and his associates discuss their experience with exchange transfusion in 160 newborn infants with hemolytic disease. As nearly all mothers of erythroblastotic children were subjected to an antepartum examination and were admitted to the hospital before delivery, it was possible to perform the exchange transfusion in nearly all cases immediately after birth, which has the advantage that the procedure goes smoothly because of the free passage through the umbilical vein. Thirty-six, or 22.5 per cent, of the infants died. Otherwise the mortality rate of icterus gravis is 73.7 per cent, or, if mild cases are included, 63.5 per cent. This difference between 63.5 and 22.5 per cent seems to indicate that the prognosis of the newborn with icterus gravis has improved considerably by the application of exchange transfusion.

Carcinoma of Breast in Castrated Women.—Dargent says that although carcinoma of the breast may occur after the natural menopause, it is a less familiar condition after castration. He encountered 32 cases (1.6 per cent) in castrated women among 2,000 cases of carcinoma of the breast. This figure is higher than that of carcinoma occurring during pregnancy (21 cases, or 1.05 per cent) or in men (16 cases, or 0.8 per cent). The longest interval before the appearance of carcinoma was in women castrated between the ages of 40 and 50. When carcinoma of the breast begins after castration, or even in spite of castration, the prognosis should be regarded as poor unless castration was performed near the time of the menopause. Routine castration after treatment of the carcinoma is not a guaranty against later manifestations of the malignant condition. Castration in advanced carcinoma of the breast has given the author 2 good temporary results (of three to thirteen months) in 10 cases in which the castration was performed surgically; of 28 cases in which castration was performed by irradiation, 4 (14.39 per cent) showed some degree of remission. Cases of highly malignant carcinoma appearing in recently castrated young women make him consider the possibility of failure of castration in similar circumstances.

Lancet, London

2:41-90 (July 9) 1949

Rôle of Cerebral Cortex in Apperception of Pain. G. W. Theobald.—p. 41.
Comparison of Anthisan (Mepyramine Maleate) and Phenergan as Histamine Antagonists. W. A. Bain, J. L. Broadbent and R. P. Warin.—p. 47.
Malleolar Fractures: An Inquiry into Their Mechanism. E. G. Herzog.—p. 52.
Vaccination with Murine Type of Tubercle Bacillus (Vole Bacillus). A. Q. Wells.—p. 53.
*Atypical Pneumonia Treated with Chloromycetin. E. J. Wood.—p. 55.

Chloramphenicol in Atypical Pneumonia.—Wood reports a man, aged 42, with primary atypical pneumonia. When the patient was 24 he had contracted pulmonary tuberculosis, which had been quiescent for some years. On the third day of his illness, when the patient's temperature showed no sign of decreasing and diaphoretics and salicylates had proved ineffective, sulfadiazine was tried, but three days later this had also proved ineffective. Roentgen examination on the eighth day revealed signs suggestive of atypical bronchopneumonic invasion. During the next four days the patient was given penicillin in divided doses, 1,000,000 units being administered during twenty-four hours. By the eleventh day it was evident that penicillin was ineffectual. The patient was then given an initial dose of six 250 mg. capsules of chloramphenicol (chloromycetin®). This was followed by 500 mg. every two hours until 4 Gm. had been taken, when the dose was reduced to 500 mg. four times daily for five further days, making the total dose 15 Gm. On the first evening of chloramphenicol treatment the subjective symptoms were less severe, and within twenty-four hours his fever

began to subside. Within forty-eight hours he expressed a desire for food; the cough became less persistent, and the sputum began to decrease. On the seventeenth day roentgen examination revealed that the bronchopneumonic patch had almost completely resolved.

South African Medical Journal, Cape Town

23:507-534 (June 25) 1949. **Partial Index**

*Tick-Bite Fever: 2 Cases Treated with Aureomycin (Lederle), New Antibiotic. J. Gear and A. L. Harington.—p. 507.
Sulpha-Masked Mastoiditis: Case. J. Fine.—p. 512.
"Open" Endotracheal Anesthesia for Tonsillectomy and Adenoidectomy in Children, with Note on Pre-Operative Medication. B. Segal.—p. 514.
Mesantoin Treatment of Epilepsy. G. M. Malan and G. G. Harrison.—p. 516.
Man's Changing Environment: Study in Human Ecology. T. W. B. Osborn.—p. 521.

Aureomycin in Tick Fever.—Gear and Harington point out that tick fever, a disease caused by Rickettsia rickettsi var. pijperi, is one of the most commonly seen infective diseases in South Africa during spring and autumn, when ticks are most prevalent. Hitherto treatment has been mainly symptomatic. In a few patients treated with para-aminobenzoic acid, as recommended for the treatment of typhus, an improvement of the patients' condition was noted but the response was not dramatic. There is therefore need for a more specific remedy. The authors treated 2 patients with aureomycin, giving a capsule containing 250 mg. orally every four hours for several days until a total up to 4 Gm. had been administered. The authors believe that the rapid recovery of these 2 patients was due to aureomycin.

Deutsche medizinische Wochenschrift, Stuttgart

74:753-784 (June 17) 1949. **Partial Index**

*Endoscopic Transpleural Approach to Thoracic Sympathetic. E. Kux.—p. 753.
Results of Recent Research on Biology of Malaria Parasites. L. Mudrow-Reichenow and W. Kikuth.—p. 759.
Value of Serologic Tests on Function of Liver. K. P. Eymer and N. Zöllner.—p. 763.
Development of Hypovitaminoses During Pregnancy. A. Hildebrandt.—p. 766.
Optic Nerve Lesions Following Immunization Against Diphtheria. P. Siegert.—p. 769.
*Specific Effect of Antiserum in Human Diphtheria. K. Kissling.—p. 775.

Endoscopic Transpleural Approach to Thoracic Sympathectomy.—Kux states that if the lung is collapsed by a preliminary pneumothorax the thoracic sympathetic system with its ramifications can be seen through the thoracoscope. It can be injected or sectioned with a cautery or a diathermy knife. This transpleural endoscopic approach has several advantages over the customary extrapleural and intrapleural surgical interventions. 1. There is practically no surgical risk. 2. The performance can be repeated; the effect of permanent exclusion can be tested by a temporary block with procaine hydrochloride. In hypertension with renal involvement the vasoconstrictors of the kidney can be excluded at the first treatment and the sympathetic nerve can be divided after the renal blood perfusion and function have been improved. 3. The section of the sympathetic nerve can be carried out with assurance and with exact localization. The new method has been used so far in four types of disorders: peptic ulcer, hypertension, angina pectoris and certain forms of hyperglycemia. The author reports 50 cases of peptic ulcer in which endoscopic sympathectomy or splanchnicotomy was done. The early results were surprising: none of these patients showed the persistent postoperative vomiting so often seen after radical vagotomy. Patients treated by vagotomy usually tolerated normal food only from three to four weeks after the operation, but those who had undergone endoscopic splanchnicotomy were able to take an unrestricted diet without any difficulties almost immediately after the operation. Pain ceases with the section of the nerve, roentgenologic evidence of the ulcer disappears usually within a few weeks and the patients are free from symptoms. The permanent results could not be ascertained, since only nine months had elapsed since the first operation of this type for peptic ulcer.

Specific Effect of Antiserum in Human Diphtheria.—Kissling shows that since the first introduction of diphtheria antiserum, that is, for over fifty years, statistical reports have appeared which throw doubt on the efficacy of the serum. These reports are based on the false premise that the efficacy of the serum can be estimated on the basis either of all patients with diphtheria that have been treated or of the total mortality rate in the course of epidemics. Authors of these reports always make the mistake of completely ignoring the premises for the therapeutic action of the serum, which von Behring repeatedly emphasized. He always stressed that his antitoxin would effect cure of diphtheria only if it is administered on the first or second day and that if the disease has advanced beyond this point the antiserum no longer can be expected to effect a cure. Reports of large numbers of cases of diphtheria nearly always revealed a 100 per cent recovery rate for those patients in whom the antiserum had been administered on the first day of the disease, while for those treated on the second day the mortality rate was small. These failures may be explained by the fact that the second day had already elapsed, since the first day is frequently disregarded by the child and the parents. Kissling cites his own experience in an epidemic of about 2,000 cases, in which only 71 patients had received the antiserum on the first day, and of these 71 none died. He takes exception to a report by Bingel, which was based on 2,919 cases but took no account of the day in the course of the disease on which the serum was administered.

Finska Läkaresällskapets Handlingar, Helsingfors

91: 133-256 (No. 2) 1948. **Partial Index**

Leukocytosis and Leukopenia. H. Hortling.—p. 133.
*Clinical Tests with Tetra-Ethyl-Ammonium Bromide Blocking of Autonomous Ganglia. G. Björkenheim.—p. 153.
Nuclear Physics and Medicine. I. H. Zilliacus.—p. 163.

Clinical Tests with Tetraethylammonium Bromide Block of Autonomic Ganglions.—Tetraethylammonium bromide in 10 per cent solution with a dosage of 4 to 10 mg. per kilogram of body weight was given intravenously to 31 patients with normal blood pressure and 20 with increased blood pressure. In the former the blood pressure decreased slightly, in the latter there was transient decrease averaging 44 mm. of mercury systolic and 22 mm. diastolic. The pulse rate was usually accelerated. The decrease in blood pressure and the pulse acceleration reached the maximum in one to three minutes. The reduction in blood pressure varied from case to case and was less in nephritic hypertension than in essential hypertension. The reduction in systolic pressure was greater in older persons than in younger persons. Björkenheim says that old age, angina pectoris and peripheral sclerosis are absolute contraindications to the use of the drug. His investigations confirm that tetraethylammonium bromide has a blocking effect on autonomic ganglions, sympathetic and parasympathetic. The paresthesias and the result of intravenous injection point to action on the peripheral sensory and motor nerves. The drug also exerts a weak, curare-like effect on striated muscles. Signs of vasodilatation are sometimes seen on intra-arterial injection. The palliative effect on pain suggests peripheral or central release of vasospasm but can also be interpreted as a local action on the nerves. Tetraethylammonium bromide may be useful in sympathectomy-resistant pain and as a preliminary test when operability in hypertension is investigated. The transient effect excludes therapeutic application in hypertension. It may be useful in acute rise of blood pressure, as in eclampsia.

Nederlandsch Tijdschrift v. Geneeskunde, Amsterdam

93:1473-1580 (May 7) 1949. **Partial Index**

Intoxication with Vitamin D. F. S. P. van Buchem.—p. 1488.
Treatment of Thrombosis and Embolism: Use of Dicumarol and Heparin. W. Hekman.—p. 1500.
*Diagnosis of Asthma, Emphysema and Silicosis by Inhalation of Aerosols Containing Vasodilators or Vasoconstrictors. J. C. Gerrits.—p. 1507.
Improvement of Schizophrenic Psychosis by Attack of Meningitis. W. Oesterreicher.—p. 1523.

Inhalation of Aerosols in Diagnosis of Asthma, Emphysema and Silicosis.—Gerrits shows that respiratory alterations which follow inhalation of an aerosol of a bronchoconstrictor substance (1 per cent of acetylcholine) can reveal the existence of a latent bronchospasm. Inhalation of a bronchodilator (1 per cent solution of isopropyl epinephrine) will show to what extent emphysema is reversible. Thus the inhalation of aerosols can be of help in arriving at a diagnosis. These aerosols cause no

changes in healthy subjects. Bronchospasm was detected in 9 of 13 patients with silicosis by means of these aerosol tests. This might explain the discrepancy in some cases of silicosis between the slight roentgenologic changes and the severe functional disturbances and shortness of breath.

Presse Médicale, Paris

57:567-582 (June 18) 1949

Spontaneous Hematoma of Cerebellum; Clinical and Therapeutic Study. J. Guillaume, R. Rogé and P. Janny.—p. 567.
*Value of Systematic Annual Roentgenographic Investigation ("Dépistage") for Detection of Pulmonary Tuberculosis. H. Brocard and G. Basset. —p. 568.
*Three Years of Systematic Annual Roentgenoscopic Investigation for Detection of Primary Infection in Adolescents. H. Werner.—p. 569.
Glycine in Treatment of Angina Pectoris. M. Audier and G. Dumon. —p. 570.

Investigation of Pulmonary Tuberculosis.—Brocard and Basset carried out systematic annual roentgenologic investigations for the detection of pulmonary tuberculosis in 25,000 workers employed with a group of industrial plants in the Paris area. The great majority of the workers were men between the ages of 15 and 70 years. Roentgen ray photographs were taken with an apparatus providing for pictures 8 by 8 cm., thus making direct reading possible. The distinctness of the pictures compared favorably with those observed on the fluorescent screen. Of the workmen studied, only 3 per thousand had Mycobacterium tuberculosis in their sputum, but in more than half of them the disease was detected by means of the roentgenograms. Suspicious pulmonary lesions were detected by this method in 3 per cent of the workers studied. This ratio shows the high incidence of latent tuberculosis, but most of these lesions were benign since only 1.4 subjects per thousand were compelled to discontinue their work. Systematic investigations for detecting pulmonary tuberculosis (dépistage) may be considered as an essential basis of industrial medicine, but a rigorous and hasty elimination of all workers with suspicious cases does not seem indispensable, particularly in a period when the sanatoriums are overcrowded.

Radioscopic Detection of Primary Infection in Adolescents.—Werner observed 89 cases of primary tuberculous infection among 353 adolescent factory workers, 243 girls and 110 boys, who submitted to repeated radiologic examination in the course of three years. Seventy-six of the 89 cases were detected by semiannual radioscopy and the remaining 13 after a series of examinations. In the absence of any acoustic signs, diagnosis must be based on changes in body weight, roentgenologic changes and conversion of skin reaction. In 2 cases the increase in the shadow of the hilus could be attributed to a descending rhinobronchitis. Any widening of the shadow of the hilus in the absence of a definite infectious antecedent should make one suspicious of tuberculosis, and the Mantoux test should be performed. Primary tuberculous infection was observed to be four times more frequent during the period of puberty than earlier. Before the admission to the factory, at the age of 14, positive skin reactions were observed twice as frequently in girls as in boys. The highest incidence of primary infection was observed in girls aged 15 and in boys aged 17. Semiannual radioscopy is the method of choice for the detection of primary tuberculous infection in adolescents. The skin reaction is a means not of detection but of late control. Radioscopy will reduce the morbidity from secondary bacillary dissemination. Of four young mothers aged 17, 3 had positive tuberculin tests before their pregnancies and did not present any incident; 1 was anergic before her pregnancy, and tuberculosis developed as a sequela of childbearing. Radioscopy should be supplemented by roentgen photographs in adolescents in highly endemic areas designated by the public health service.

Revista Española de Pediatría, Zaragoza

5:157-306 (March-April) 1949. **Partial Index**

*Tuberculous Meningitis: Streptomycin Therapy. M. Suárez.—p. 262.

Streptomycin in Tuberculous Meningitis.—Suárez reports 28 cases of tuberculous meningitis treated with streptomycin. Tolerance of the drug appeared to be greater in infancy than in childhood. Best results were obtained with early treatment. One milligram intraspinally and 20 mg. intramuscularly were given on alternate days. A course of treatment lasted one month and was resumed after a period which depended on the clinical course and the disappearance of bacilli from the cerebrospinal fluid. The treatment should be continued for one or two years with a minimal period of hospitalization of eight months. Streptomycin in doses used by the author was well tolerated. Prevention of acquired resistance of bacilli to the drug is accomplished by following the scheme of small doses, courses of treatment of short duration and proper pauses between the courses. When the treatment was begun after the tenth day of the disease, the mortality rate was high. When the drug was given in the course of the first five days, better results were obtained. Five of the patients who had entered the hospital in the second week of the disease died within the first two weeks of hospitalization. Of the remaining 23 patients, 9 died after transient improvement which lasted two to seven months. Fourteen patients got well without sequels. The follow-up period in this group is over two years. The 2 patients who got well but presented sequels, mental deficiency in 1 and meningoencephalitis in the other, had a grave type of the disease.

Schweizerische medizinische Wochenschrift, Basel

79:589-612 (July 2) 1949. **Partial Index**

Vaccination of School Children with BCG: Preliminary Report on Frequency of Tuberculous Infection in School Children, Aged 15 and 16. G. Guye.—p. 589.
*Electroshock Therapy Under Anesthesia With and Without Curare. P. Kielholz and J. Heuscher.—p. 592.
Disturbances in Blood Coagulation by Beta and Gamma Globulins. E. Lüscher and A. Labhart.—p. 598.
Is Bronchial Adenoma a Benign Tumor? R. H. Jenny.—p. 604.

Electroshock Therapy Under Anesthesia, With and Without Curare.—Kielholz and Heuscher have given 120 electroshock treatments with the patient under thiopental (pentothal®) anesthesia, occasionally combined with curare. When only thiopental is used, 1 cc. of a 5 per cent solution is injected intravenously each minute, until the patient fails to react to a call. Only then is the electroshock apparatus brought into the room and the shock treatment given in the usual manner. After the shock the patient is kept under observation; he is not roused but wakens spontaneously. If curare is administered in addition, the thiopental solution is given first until the necessary depth of anesthesia has been obtained. Then the computed quantity of curare is injected through the same needle within two minutes. After the lapse of an additional three to four minutes (maximal hypotensive effect of curare), electroshock is induced. Immediately after the shock, 1 cc. of prostigmine is injected into the vein and the patient is watched until he wakens. The authors say that the method which they use resembles that described by Brody. They stress that the combination with anesthesia makes it possible to employ electroshock therapy even in patients who show extreme anxiety and in those who would reject it because of fear. Shock treatment with the patient under anesthesia can be given even to those over 50 years of age.

79:613-636 (July 9) 1949. **Partial Index**

Significance of Rh/rh Blood Group Systems for Practical Medicine. P. Dahr.—p. 613.
*Eosinophilic Granuloma of Bone. B. Walthard and A. Zuppinger. —p. 618.
Renal Function in Myxedema. H. Zondek and G. Wolfsohn.—p. 623.
Antihistamine Action of Tannin. D. Gyüre and A. Kovács.—p. 624.
Treatment of Tuberculous Empyema by Means of Para-Amino-Salicylic Acid (PAS). A. Gilliard.—p. 625.

Eosinophilic Granuloma of Bone.—Walthard and Zuppinger report 8 patients with a new clinical entity, designated by Otani and Ehrlich and by Lichtenstein and Jaffe as eosinophilic granuloma of bone. Five of their 8 patients were less than 10 years of age and 3 were in the second decade of life. This age incidence corresponds with that in other reports, but the fact that the sex distribution was even (4 and 4) differs from most reports which indicate a greater incidence in males. Cranium and ribs are the preferred localization of this bone lesion. Roentgenography reveals a rapidly developing osteolytic lesion, in which marginal sclerosis is either slight or completely missing. A definite diagnosis requires microscopic examination,

which reveals a reticulating ground tissue with round or oval cells. Eosinophilic leukocytes are either diffusely disseminated or are arranged in foci-like microabscesses. Multinuclear giant cells are loosely arranged in the protoplasm and often show phagocytosis. Macrophages contain nuclear debris, eosinophilic leukocytes and hemosiderin. There are moderate numbers of lymphocytes, plasma cells and neutrophilic leukocytes, also hemorrhagic foci and small foci of necrosis. The walls of the blood vessels are often thickened and infiltrated with eosinophilic leukocytes. There is destruction of bone, even formation of sequestrums; and there is periosteal formation of new bone with numerous osteoclasts. Charcot-Leyden crystals are found chiefly in the necrotic foci. Foam cells are found, and there is transformation of granulation to connective tissue. The lesion heals following surgical removal of the granulations. Roentgen irradiation may also effect a cure. If the granulomas are located close to joints, radical surgery is not advisable. If the lesion is located in the middle ear, a combination of surgical measures and roentgen irradiation gives the best results. Although spontaneous cure is possible, it is not advisable to leave a lesion alone because large defects in the bone may lead to spontaneous fractures or functional disturbances.

Semaine des Hôpitaux de Paris

25:1877-1910 (June 14) 1949. **Partial Index**

Indications for Lumbar Puncture in Course of Treatment of Miliary Tuberculosis. J. Tapie, J. Laporte and Monnier.—p. 1877.
*Phthisiogenetic Primary Infection. H. Brocard.—p. 1881.
*Tuberculosis of Endometrium; Typical and Atypical Histologic Aspects in 14 Cases. J. Bret, B. Duperrat and J. Tronc.—p. 1893.

Primary Phthisiogenetic Infection.—According to Brocard, the term primary phthisiogenetic infection is to be applied to cases in which terminal pulmonary tuberculosis follows the primary infection at a short interval of not more than three years. A reaction of the serous membranes, particularly of the pleura, may be the intermediary between the primary and the secondary infection. The presence or absence of this intermediary and the patent or latent character of the primary infection create various clinical types of the disease. One may distinguish an abortive, common and malignant primary phthisiogenetic infection. Primary infection is predominant in adolescents and young adults between the ages of 15 and 25. Certain races, such as the African Negroes and the Algerian Moslems, seem to have a specific susceptibility to the malignant types. Differentiation between a focus of primary infection and a focus of secondary tuberculous lesion presents an important diagnostic problem which may be solved only by a prolonged follow-up of the patient. The increased incidence of primary tuberculous infection is due to the fact that the average age at which the primary infection occurs has drawn nearer to the phthisiogenetic age. Strict rest may prevent the primary infection from becoming phthisiogenetic, but the best method of prophylaxis is that of preventing the primary infection by avoiding exposure of young anergic adults to massive contamination and by practicing BCG vaccination.

Tuberculosis of Endometrium.—Bret and co-workers report 14 women between the ages of 25 and 51 with tuberculosis of the endometrium. Sterility was the major symptom in the great majority of the cases. The general condition of the patients was satisfactory. History revealed familial or personal tuberculous antecedents in only 5 patients. Biopsy is the only means of establishing the diagnosis. Typical tuberculous lesions such as tubercles with giant cells and with a tendency to necrosis or suppuration, and tubercles formed by masses of epithelioid cells of Langhans type were observed in 9 patients, 5 of whom had tuberculous antecedents. In the remaining 5 patients the biopsy revealed atypical lesions such as tubercles with some epithelioid cells surrounded by lymphocytes or lymphoid cells suggesting various etiologic concepts. Tuberculosis of the endometrium occurs frequently without any accompanying sign and without tuberculous antecedents. Two patients were given calcium and benzyl cinnamate; menstruation recurred but biopsies carried out later did not reveal any changes in the lesions. Hysterectomy is not advisable, except in the rare cases in which the adnexa are involved. It is important to prevent pregnancy because of the grave postpartum complications and because there is no assurance that the infant will be normal. Symptomatic hormonal medication is contraindicated.

Wiener klinische Wochenschrift, Vienna

61:321-336 (May 27) 1949. **Partial Index**

Psychic Hygiene. E. Stransky.—p. 321.
*Contribution to Early Diagnosis of Periarteritis Nodosa by Means of Sternal Puncture. O. Griesbacher.—p. 325.
Life Threatening Accident in Transfusion, After Intravenous Inoculation of Malaria Blood of Disparate Blood Groups. J. Konard and F. J. Holzer.—p. 330.
Periurethritis Due to Foreign Body (Grass Leaf). H. Kellner.—p. 332.

Periarteritis Nodosa.—Griesbacher reports 4 cases of periarteritis nodosa in which relatively early clinical diagnosis could be established by microscopic examination. The patients had marasmus, unexplained rises in temperature, renal hypertension and polyneuritis. Repeated sternal puncture revealed eosinophilia, increase in reticulocytes and lymphopenia. These characteristic changes in the bone marrow and the moderate eosinophilia correspond to the concept that in periarteritis nodosa there is a hyperergic-allergic process in the course of a generally infectious and toxic or septic disease. The disparity between the number of undiagnosed and in vivo diagnosed conditions may be explained by the confusing polymorphism of symptoms and by the relatively rare occurrence of the main symptom, i. e., of the nodules. In cases which are clinically suspect, exploratory excision of the abdominal skin should be repeated at intervals of four to six weeks; with this technic the nodules might be demonstrated on microscopic examination, as it happened in one of the author's cases. The changes in the bone marrow observed by the author may be added to the cardinal symptoms of the disease.

61:337-352 (June 3) 1949. **Partial Index**

Rapid Agglutination Tests in Patients with Typhoid and Paratyphoid Fever. J. Möse.—p. 341.
*Penicillin Treatment of Acute Osteomyelitis in Children. S. Szücs and J. Ladányi.—p. 343.

Penicillin in Acute Osteomyelitis.—Szücs and Ladányi treated acute osteomyelitis in 39 children with sulfonamide drugs alone and in 21 children with sulfonamide drugs and penicillin. In combined sulfonamide and penicillin treatment 5,000 to 20,000 units of penicillin were given every three hours by the intramuscular route. Immobilization of the affected member was carried out by means of a plaster of paris cast. Subperiosteal abscesses were opened after twenty-four hours' treatment with penicillin; drainage was carried out, and local treatment with penicillin was practiced occasionally. Because of the small supply of penicillin the total dose of penicillin varied from 400,000 to 800,000 units, except for a few instances in which 2 to 3 million units were given. In 10 of the 39 patients treated with sulfonamide drugs alone trepanation or subperiosteal resection had to be performed, but not a single major bone operation was required in the group treated with sulfonamide drugs and penicillin. Complete recovery was obtained with sulfonamide therapy alone in only 9 of the 39 patients; complete recovery resulted in 13 of the 21 patients receiving penicillin in addition. Five of the patients treated with sulfonamide drugs alone died. Of the 4 deaths in the penicillin-treated group, 1 occurred in an infant aged 1 month with congenital syphilis and 1 in a marantic infant aged 3 months who could be given only 100,000 units of penicillin. The third and fourth death occurred within twenty-four hours in 2 children aged 5 and 13 years in whom the osteomylitis ran a fulminant, septic course. Roentgenologic examination of the patients treated with penicillin showed that osteitic foci in the bone tissue were either absent or less extensive than such foci in patients not treated with penicillin. Sequestration occurred in 14 of the 39 patients treated with sulfonamide drugs alone but in only 3 of the 21 patients treated with penicillin. The duration of the disease was more than two months in 19 of the 39 patients treated with sulfonamide drugs alone and less than two months in all 21 patients treated with penicillin. Improvement in diagnosis, well timed treatment by a specialist and administration of penicillin have brought about a decisive change in the management of osteomyelitis.

Book Notices

The Principles and Practice of Rectal Surgery. By William B. Gabriel, M.S., F.R.C.S., Surgeon to St. Mark's Hospital for Cancer, Fistula, and Other Diseases of the Rectum, London. Fourth edition. Cloth. Price, $14.50. Pp. 508, with 289 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1948.

This book, which consists of 18 chapters, 508 pages and 278 illustrations with 11 fine color plates, has been compiled from the author's vast experience and deals with the diagnosis and treatment of diseases of the colon and anus. It contains many details of technic and statistics which have been brought up to date and takes into account the increase in knowledge of rectal diseases which has been made since the publication of the last edition of this book. However, some of the methods described are obsolete, particularly those pertaining to the position of the patient, the technic of proctoscopy and postoperative care. In general, however, methods whereby the best results are likely to be obtained have been selected. The author, who is well known, has provided a complete account of rectal surgery which possesses value for all of those physicians who are interested in diseases of the colon. This is a good book from the pen of a surgeon of integrity and experience.

The Story of Medicine. By Joseph Garland, M.D. Cloth. Price, $2.75. Pp. 259, with 23 illustrations by Erwin H. Austin. Houghton Mifflin Company, 2 Park St., Boston 7, 1949.

This is a brief but comprehensive and well written résumé of medical history from the medicine man of ancient times to the allegedly vanishing family doctor of today. The author envisions for the latter a continuing place in our community life because the "humane and kindly and understanding practice of medicine could not proceed without him. In the laboratory and in the great teaching hospital we may find the science of medicine, but in the family doctor's office or at the bedside in time of suffering we will find its art."

The book is intended for 12 year old and young adult readers and is admirably suited for them. The book is simply written with clarity, precision and pleasant informality. It should find particular usefulness as a reference book in the junior high and high school libraries and in public libraries, since it is not too elementary to be useful even for adults as a rapid survey of medical history and progress.

Psychiatric Examination of the School Child. By Muriel Barton Hall, M.D., Honorary Assistant Medical Psychologist, Royal Liverpool United Hospital. Cloth. Price, $4.50. Pp. 368. William Wood & Co., Mount Royal and Guilford Aves., Baltimore 2, 1947.

This excellent book, though not a textbook, provides principles and clinical material necessary for clinical examination and psychiatric diagnosis of children. Although intended for the general practitioner, the book is written in sufficiently nontechnical language to be useful to educators, social workers and others concerned with child welfare and care. It represents the experience of a psychiatrist long associated with child guidance. Its purpose is to provide orientation and guidance, which it does admirably. The specialist will need more explicit and detailed clinical discussion, and the author gives appropriate indication of further sources in an adequate bibliography. A brief but admirable discussion of legal measures now in force in England, pertaining to the status and care of the handicapped and delinquent child, is of special interest.

Atomic Energy Year Book. Edited by John Tutin, D.Sc. Cloth. Price, $3.85. Pp. 237, with illustrations. Prentice-Hall, Inc., 70 5th Ave., New York 11, 1949.

Progress in the investigation of atomic energy is so rapid and much of it so carefully concealed that few scientists are aware of all that is being accomplished. This purports to be the first edition of an annual volume on atomic energy. It begins with a history of nuclear science and a description of activities in various phases. The seventh chapter provides a survey of developments in the field of atomic energy, and the eighth chapter deals with the legal aspects. Of special interest to the medical profession are chapters 10, 11 and 12, which are concerned with human welfare and radioisotopes. The appendixes provide data in tabular form, a list of national scientific academies and reference to the important books on the subject.

Factors Regulating Blood Pressure. Edited by B. W. Zweifach and Ephraim Shorr. Transactions of the Second Conference, January 8-9, 1948, New York, N. Y. Loose-Leaf. Paper. Price, $2.75. Pp. 170, with illustrations. Josiah Macy Jr. Foundation, 565 Park Ave., New York 21, [n. d.].

This publication is a series of reports of work in progress by outstanding investigators in this country. It is primarily concerned with the further study of the humoral and chemical factors in the regulation of blood pressure. These studies are directed toward a clarification of the mechanisms involved in the production of experimental renal hypertension and of so-called essential and malignant hypertension in man.

The book contains nine papers which were read at the Second Conference on "Factors Regulating Blood Pressure," sponsored by Josiah Macy Jr. Foundation. Participating in the conference were Frank Fremont-Smith, medical director of the foundation, and Harry Goldblatt, chairman of the conference. The other participants were Ephraim Shorr, Stanley E. Bradley, Lewis Dexter, William Dock, Thomas F. Dougherty, F. W. Dunihue, William Goldring, Arthur Grollman, O. M. Helmer, Robert Loeb, Abraham Mazur, Eric Ogden, Jean Oliver, Norman S. Olsen, Irvine H. Page, George Perera, Henry A. Schroder, Hans Selye, Robert E. Shipley, Nathan Shock, George E. Wakerlin and Benjamin W. Zweifach.

The papers presented by this impressive list of investigators are rather complex and are fully discussed by the various participants. The publication is of great value to those concerned with experimental hypertension and its possible relation to hypertension in man. It brings up to date the vast amount of investigative work which has been carried out during the past fifteen years. It is apparent from this publication that a great deal of further investigation is needed before the significance of humoral and chemical mediators of hypertension can be established.

Those who are particularly interested in this important subject should study these valuable reports in detail.

Personal Adjustment in Old Age. By Ruth Shonle Cavan, Assistant Professor of Sociology, Rockford College, Rockford, Ill., Ernest W. Burgess, Chairman, Department of Sociology, the University of Chicago, Robert J. Havighurst, Executive Secretary, Committee on Human Development, the University of Chicago, and Herbert Goldhamer, Associate Professor of Sociology, the University of Chicago. Cloth. Price, $2.95. Pp. 204. Science Research Associates, Inc., 6755 N. Clark St., Chicago 26, 1949.

The literature of gerontology, the science of aging, is growing rapidly in all of the three major categories into which this immense field is divided: the biology of senescence, clinical geriatric medicine and sociologic gerontology. The present volume is a report of studies made by a group of sociologists from the University of Chicago. According to the authors the book has three purposes: to define and analyze the problems of personal adjustment in old age, to present data from census reports anent the magnitude of the problem and information gleaned from a special study of nearly 3,000 old persons made by the group and to describe and evaluate two questionnaire instruments (Inventory of Activities and Index of Attitudes) employed in making the survey.

The volume contains many "facts," but some of them are of questionable validity; the data are insufficiently correlated and inadequately summarized. Unfortunately none of the participants of this study were physicians, biologists or trained psychologists or psychiatrists. The lack of clinical and psychiatric background is manifested repeatedly; for example, in the assumption that persons who failed to adjust emotionally to old age had been well adjusted and adequately integrated during their early and middle maturity. Such an assumption is obviously unwarranted and totally ignores the fact that personality problems and lack of emotional homeostasis continue from one phase of the life span into the next.

The role of somatic health as a factor in adjustment to old age is given some consideration, but not enough, for the status of health was evaluated not by clinical examinations but on the basis of the reports received from the two questionnaires. The obvious fact that the aged arteriosclerotic person is not aware of his cerebral deterioration and/or the cause thereof is ignored. Read critically the book offers much to those interested in the social medicine of old age; read with full acceptance of all its statements it may be sadly misleading. Clearly the conclusions

are drawn from "paper people," not from living, frightened, struggling, sick, old men and women.

This volume, with its faults as well as its many virtues, emphasizes the urgent need for intimate cooperation between the so-called social sciences and clinical medicine. Medicine, both as a science and in practice, does not stand alone in splendid aloofness, isolated in space, but operates in a social medium in which people are constantly being affected by their interpersonal relationships and economic and physical environments. At the same time they affect their environment by their existence and behavior. Heretofore clinicians have paid far too little attention to the sociologic aspects of their work. In this connection, a recent volume (Galdston, I.: Social Medicine: Its Derivation and Objectives, New York, The Commonwealth Fund, 1949) is recommended reading. Perhaps some of the relative disrepute which handicaps sociologists in their search for funds for research studies stems from their ignorance of individual human behavior, clinical medicine, psychology and psychiatry. Departments of sociology need physicians on their staffs; medical schools need sociologists to assist in the maintenance of broad perspectives. Understanding is distorted by either too broad or too narrow a point of view. Medicine has been concerned almost solely with disease. The common problems of so-called normal persons is an area of medical investigation which has thus far received far too little attention, for optimum health is something more than the absence of disease. All concerned with the problems of aging can profit by the study of reports of sociologic as well as clinical investigations.

Pediatrics and the Emotional Needs of the Child as Discussed by Pediatricians and Psychiatrists at Hershey, Pennsylvania, March 6-8, 1947. Edited by Helen L. Witmer. Paper. Price, $1.50. Pp. 180, with 16 illustrations. Commonwealth Fund, 41 E. 57th St., New York 22; Oxford University Press, Amen House, Warwick Square, London, E.C.4, 1948.

This is a well edited summary of discussions held in the spring of 1947 at Hershey, Pa. Participants were leading pediatrists, child psychiatrists and social workers. In the three day session, as guests of the Commonwealth Fund, the participants surveyed the relationship of the pediatrician to the field of mental health, developments in departments of pediatrics bearing on this relationship and the required steps to provide the teaching and training necessary to equip the pediatrician for adequate functioning in this field. It is apparent in this discussion that the pediatrician of today rapidly is becoming better oriented in the psychologic aspects of his specialty and that pediatrics, always a preventive medical specialty, now comes more into its own as a discipline designed to maintain and preserve good mental health as well as sound physical development for the growing child. The refreshing and encouraging exchange of views between pediatrician, psychiatrist and social worker as presented in this report would not have been possible a few years ago. Of particular interest are the discussions of the efforts of teaching centers to provide training for psychiatrically oriented pediatricians. There is little precedent, much divergence of view and practice in this field now being carefully explored in an increasing number of teaching centers.

This work should be of interest to all pediatricians and is a must for all who bear responsibility for training and teaching of pediatricians.

Industrial Health Department Functions and Relationships: A Quarter of a Century of Progress in Industrial Health Administration. By C. O. Sappington, M.D., Dr.P.H. Medical Series: Bulletin No. VIII. Paper. Pp. 98, with illustrations. Industrial Hygiene Foundation, 4400 5th Ave., Pittsburgh 13, 1948.

The most encouraging revelation in this two year study sponsored by the Industrial Hygiene Foundation of Pittsburgh is the considerable gains which have been made since Selby's similar review for the Public Health Service in 1919. The subtitle of the brochure is "A Quarter of a Century of Progress in Industrial Health Administration." Present conditions in 278 plants employing 1,180,551 workers were analyzed, with particular attention to personnel, physical plant, functions, intramural and extramural relations, reasons for maintenance and miscellaneous data relating to records, floor plans, procedure and the like. As in many similar studies, it is again demonstrated that large plants are in a position to avail themselves of competent and reasonably complete health services and that extension to small concerns is still the great unsolved problem. Much of the data in text and tables supports this conclusion. Sappington's suggestions for further improvement in the broad industrial health picture are all pointed in that direction and depend fundamentally on willingness of management to underwrite and qualified physicians to supply adequate health programs in plants with under 1,000 employees.

With respect to plants of all sizes, he urges more extensive use of periodic physical examinations, industrial hygiene facilities, dental services and health education. A physician or plant manager considering the installation of a medical program will find this compilation most helpful.

Health Education in Schools. By Jesse Feiring Williams, M.D., Sc.D., Emeritus Professor of Physical Education, Teachers College, Columbia University, New York, and Ruth Abernathy, Ph.D., Associate Professor of Physical and Health Education, University of Texas, Austin. Cloth. Price, $3.50. Pp. 316, with 23 illustrations. The Ronald Press Company, 15 E. 26th St., New York 10, 1949.

This book is intended for students and teacher-training institutions and for teachers in active teaching situations. It gives a complete background for health teaching, including a basic orientation, discussions of health, personnel and healthful living and an exposition of the basic nature of the child. School health services, such as health examinations, disease prevention and the correction of physical abnormalities, are described, as is the role of official and nonofficial organizations in health education. Among the professional agencies, the American Medical Association, state medical societies and county medical societies are recognized. The teaching of health as such, the relation of health teaching to the curriculum as a whole and methods of instruction in health are likewise discussed with completeness and clarity. The chapter on evaluation is of particular interest, since evaluation is admittedly the most difficult phase of health education. In this chapter various testing methods are discussed, together with general criteria which should be useful to the teacher attempting to evaluate his own program. Considerable attention is given to the health of the teacher, which is all too often overlooked. Each chapter contains a list of questions for discussion and review. The book can be highly recommended as a test for teacher-training institutions and as an ever handy reference book on the desk of the teacher in service.

The Natural History of Mosquitoes. By Marston Bates. Cloth. Price, $5. Pp. 379, with 25 illustrations. The Macmillan Company, 60 5th Ave., New York 11, 1949.

From the vast literature on the history and biology of mosquitoes Dr. Bates has attempted to compile the known facts concerning mosquito behavior as well as to point out the needed studies. The material included is frequently classified under the discipline of ecology and physiology. The first part of the book is organized around the life history of the mosquito, taking up in turn the characteristics of behavior, physiology and environmental relations of the adult, larval and pupal stages.

The medical man will be particularly interested in chapter 14, which deals with mosquitoes as vectors of viruses. The relations of these insects as transmitters of yellow fever; dengue; Venezuelan, western and eastern equine encephalitis; St. Louis and Japanese B. encephalitis; West Nile virus; lymphocytic choriomeningitis, and Rift Valley fever are discussed. In addition, several other viruses have been recovered from mosquitoes in nature. Equine infectious anemia, fowlpox and rabbit myxoma are virus diseases of animals which are transmitted by mosquitoes. The following chapter deals with mosquitoes as vectors of the various species of malaria parasites. Chapter 19 discusses technics in mosquito study and provides useful data for those actively engaged in mosquito research. A bibliography, 46 pages in length, concludes the text. The author has pointed out that this is probably only one fifth of the literature, but the titles which have been cited include the important reviews and will serve as keys to the remainder of the publications on this general subject.

Sixteen pages of plates depicting a wide variety of mosquito breeding places as well as technics utilized in the collection or

rearing of mosquitoes form an attractive and useful addition to the text.

For more than a decade the author has been a member of the staff of the International Health Division of the Rockefeller Foundation actively engaged in the study of the natural history of mosquitoes in the Mediterranean region, South and Central America, as well as in making visits to many other parts of the globe where research on malaria and yellow fever has been in progress. The present digest of the literature combined with his own enormous experience has resulted in the preparation of a book which should be of particular interest to workers in the field of biology, medicine and public health.

The 1948 Year Book of Neurology, Psychiatry and Neurosurgery. Neurology: Edited by Hans H. Reese, M.D., Professor of Neurology and Psychiatry, University of Wisconsin Medical School, and Mabel G. Masten, M.D., Associate Professor of Neuropsychiatry, University of Wisconsin Medical School, Madison, Wis. Psychiatry: Edited by Nolan D. C. Lewis, M.D., Director, New York State Psychiatric Institute and Hospital, New York. Neurosurgery: Edited by Percival Bailey, M.D., Professor of Neurology and Neurological Surgery, University of Illinois, Chicago. Cloth. Price, $5. Pp. 750, with 140 illustrations. The Year Book Publishers, Inc., 304 S. Dearborn St., Chicago 4, 1949.

This annual publication is one of thirteen comprising the practical medicine series. It contains 750 pages consisting of reviews of neurologic entities (249 pages), psychiatric diseases (223 pages) and neurosurgical procedures (239 pages). This book is primarily a review of some of the studies appearing in print relative to neurology, psychiatry and neurosurgery. This volume at present can be useful for the general practitioner in that some of the important nervous system diseases are reviewed. Unfortunately, this type of book only surveys various printed forms of endeavor without "pro and con" discussions. The excellent monthly publication of section VIII of "Excerpta Medica" has replaced this "Year Book" for specialists. The "Excerpta Medica" publishes a review of every article in the entire world dealing with neurology, psychiatry, neurosurgery and therapy. In the "Year Book" the neurosurgical section is relatively new and should be of some help to all physicians and surgeons interested in surgery. New technics are written and also are given some personal expressions from the author. This is valuable. Transorbital lobotomy, repair of cranial defects, epilepsy, hypertension, intervertebral disk and peripheral nerve injuries are reviewed. The twenty queries on the front flap of the book cover are out of order in a book of this kind. Certainly the giving of easy methods of examination of the patient would be practical and useful to the physician, who then would develop a technic or method of examining his patients and carrying out the necessary treatment.

Encyclopedia of Criminology. Edited by Vernon C. Branham, M.D., Chief, Outpatient Section, Neuropsychiatric Division, Veterans Administration, and Samuel B. Kutash, Ph.D., Chief Clinical Psychologist, Newark Mental Hygiene Clinic, Veterans Administration. Cloth. Price, $12. Pp. 527. The Philosophical Library, Inc., 15 E. 40th St., New York 16, 1949.

Many contributors have joined with the editors to produce here a most unusual work. In alphabetic order with many references to literature, the authors provide brief discussions of all of the subjects that can be grouped under the heading criminology. The allocation of space indicates the lack of a carefully organized plan, since some subjects, like the alcoholic criminal, occupy many pages and others apparently of equal importance are dismissed with a paragraph. The section on criminal law and procedure occupies a dozen pages and expert testimony six. The bibliography on expert testimony is especially well selected. The section on insurance crimes is also given seven or eight pages, whereas the intelligence quotient receives less than one. Altogether, however, this is a comprehensive useful work of reference.

One is surprised by the heading "white collar criminal" under the letter W; this discussion includes, strangely, the indictments of great corporations, with the apparent implication that all have violated the law. The author does not believe that redistribution of wealth would prevent such "crimes." The term constitutional psychopathic inferior does not appear in the book or in the index, although this is one of the most difficult factors to deal with in crime control.

A Doctor Talks to Teen-Agers: A Psychiatrist's Advice to Youth. By William S. Sadler, M.D., F.A.P.A., Consulting Psychiatrist, Columbus Hospital, Chicago. Cloth. Price, $4. Pp. 379. C. V. Mosby Co., 3207 Washington Blvd., St. Louis 3, 1948.

This book bears the subtitle, "A Psychiatrist's Advice to Youth." Inspirational and fatherly in tone, it deals with life adjustments in general, sexual problems and the problems of courtship and marriage. The author attempts to reproduce in these pages the atmosphere of the consultation room in which he has talked with young persons who have sought his advice or guidance.

The author's purpose and his encouraging, friendly approach are most commendable. The task he has set himself, however, is difficult. The teen-age range is rather broad, and this book is suitable only for the older and more mature of the teen-agers. The title should be modified to indicate this, as there is little question that the book would be more disturbing than helpful to the younger teen-agers not ready to deal with some of the material presented. Much of the material should be definitely reserved for a book on marriage counseling.

The author raises, indirectly, important technical questions relating to treatment and counseling of young persons by psychiatrists. He refers to the young girl who sees a psychoanalyst a few times, learns about her sexual "repressions," abandons treatment and repressions for a period and is finally helped to more stable perspective through the author's counseling. The author utilizes this situation for a dissertation on the value of retaining "repression." Such a discussion does not make much sense in the present book, particularly since the author is himself confused. Maintenance of self control and self discipline is always aided by the mechanism of repression. It is the technical task of the psychiatrist to understand whether, in the given instance, such defense serves a healthy or unhealthy purpose, and his dealing with the patient will vary accordingly.

This book, if recommended at all, should be recommended only for persons of college age.

Janos: The Story of a Doctor. By John Plesch. Translated by Edward Fitzgerald. Cloth. Price, $5. Pp. 579, with 37 illustrations. A. A. Wyn, Inc., Publishers, 23 W. 47th St., New York 19, 1949.

The title of the book is the Hungarian name for John, the name by which Prof. John Plesch is known to his intimates. Truly here is an informative and exceedingly fascinating autobiography. The doctor has been associated in his life with many of the great and near great in history, with scientists, royalty, musicians, artists and statesmen.

The book is divided into three parts, the first devoted to science, politics and personalities and the second to the theater, art, music and England. The third part, which is an appendix, is entitled "A Doctor's Dialogues" and seems to have the type of material that in the United States would be included in health columns. Most of it is in the form of questions and answers. The doctor believes that women during times of plenty cultivate voluptuous figures which men admire in such circumstances, whereas in economic and other crises, the women try for the slender figure. He believes that it is not advisable to meddle with constitutional tendencies.

Among the notables who wander through these pages are Elizabeth Bergner, Reinhardt, Kreisler, Toscanini and Marlene Dietrich. The author tells his story with numerous discussions of his philosophic points of view. The volume merits a place in any collection of important medical autobiography because its author is a man of talent and the history of his life is well worth telling.

Man-Made Plague: A Primer on Neurosis. By William G. Niederland, M.D., Medical Director, Sanatorium Rheinburg, Lake Constance, Switzerland. Cloth. Price, $3.50. Pp. 304. Renbayle House, 1165 Broadway, New York 1, 1948.

Neurosis is the popular disease of our present generation. This work purports to present a definition of neurosis, its signs and symptoms, its causes, its prevention and its methods of treatment. The book leans heavily on the writings of others, notably Alvarez. This volume presents a great deal of material easily available elsewhere and presented elsewhere in a far more distinguished manner.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

ANTIVENIN, TETANUS ANTITOXIN AND SILVER NITRATE

To the Editor:—**Please evaluate the use of antivenin in the treatment of snake bite. Has its value been scientifically demonstrated? Please do the same for tetanus antitoxin in the treatment of wounds and silver nitrate (Credé) for the newborn infant's eyes. I believe that these three measures should be subjected to critical evaluation because of their reputation and legal implications and the tendency of the medical profession to hold onto established dictums in the absence of satisfactory evaluation.**

W. E. Lockhart, M.D., Alpine, Texas.

ANSWER.—Human poisoning from snake bite is observed only occasionally and then usually under conditions that make it difficult to obtain a critical analysis of antivenin therapy. Antivenins for snake bite can be standardized for potency in animals and can be prepared to provide sufficient antibody in each recommended dose to neutralize the average estimated quantity of venom in a single snake bite. It is generally conceded, however, that the effects of venom that may be introduced by a single bite are influenced by the size of the snake and the amount of venom present at the time of the bite. If a snake has recently bitten another victim the amount of venom injected into the second victim will be less. The size of the person or animal bitten and the time elapsing between the bite and injection of serum also are influencing factors. Nevertheless, many agree that antivenins are of some value and that the value increases with the promptness of administration: usually half the recommended dose (doubled for children) is given locally about the wound, half intramuscularly or intravenously, depending on the urgency. Prompt first aid measures are essential and include application of a tourniquet until serum is administered and local suction of the wound to remove as much poison as possible. Local incisions or application of chemicals are not advised. A discussion of antivenin therapy in Southwestern United States may be found in "Modern Treatment of the Bites and Stings of Small Desert Animals" (*Arizona Med.* **5**:54 [July] 1948). The Council on Pharmacy and Chemistry has accepted a brand of antivenin crotalus (North American antisnake-bite serum).

Tetanus antitoxin has long been established as valuable in the prevention of tetanus, and most authorities today are agreed that it should be administered promptly with adequate doses in all recent exposures when active immunity has not been induced with tetanus toxoid. If immunity has been induced, a stimulating dose of the toxoid should be given to avoid the risk of serum reaction. The value of antitoxin for the treatment of frank tetanus is much less certain. Here again, it is difficult to evaluate its possible benefit because of variables from patient to patient, but the present state of knowledge indicates that the use of antitoxin should be considered secondary to the elimination of the anaerobic focus of organisms in the contaminated wound by excision of all debris and foreign body material as well as devitalized tissue. When this is not done or inadequately done, patients are more likely to succumb to tetanus despite the adequate use of antitoxin. Because of possible spinal cord injury due to antitoxin, intrathecal injection should be abandoned, and it should not otherwise be employed without regard for the possible consequences of sensitivity to horse serum. The question of adequate dosage has been previously discussed (Spaeth, R.: Immunization Against Tetanus, J. A. M. A. **132:** 667 [Nov. 16] 1946).

The question of the value or possible harm in the use of silver nitrate as a routine prophylactic for gonorrheal ophthalmia in newborn infants is still unsettled, despite the recent attention that has been focused on this problem in connection with studies employing penicillin in place of the Credé method.

An editorial in THE JOURNAL (May 28, 1949, p. 410) indicates that on July 20, 1948, the Council of the American Academy of Ophthalmology and Otolaryngology and the Section of Ophthalmology of the American Medical Association approved a report of a joint committee which concluded that the incomplete state of our knowledge on this subject did not justify any change in existing legislation where this now specifies the use of silver nitrate. Investigations to eventually settle this important question are now in progress, but lengthy observations will be required for statistical comparison of the Credé method with modern forms of prophylaxis.

EFFECT OF HIGH ALTITUDE

To the Editor:—**What effect may a change of habitat from sea level to an altitude of 6,500 feet be expected to have on one's general health? In what types of cardiovascular or other diseases is such a move contraindicated? Discuss indications and advisability of recommending such a change for a fairly large group of office workers.**

Eugene A. Stanchi, M.D., Freeport, N. Y.

ANSWER.—If a person is acutely exposed to an altitude of 6,500 feet, the physiologic changes observed are those of a mild arterial anoxemia. Qualitatively, the following events occur: The reduction in barometric pressure reduces the oxygen pressure of inspired air which in turn results in a fall in alveolar and arterial oxygen pressure. The latter reduces the arterial oxygen saturation and also stimulates breathing via the aortic and carotid chemoceptors. The increased ventilation blows off carbon dioxide and produces a respiratory alkalosis. Thus, on acute exposure, the final picture is one of arterial anoxemia and uncompensated respiratory alkalosis. Quantitatively, however, as shown in the table, all of these changes are so small at 6,500 feet that no symptoms of any sort are usually experienced. No increase in cardiac output occurs.

If one remains at altitude, certain events characteristic of acclimatization occur which require about a month for completion. At 6,500 feet, the most important changes consist of a gradually increasing ventilation with a further lowering of alveolar carbon dioxide pressure and a corresponding rise in oxygen pressure. In spite of the falling carbon dioxide pressure, however, the arterial p_H returns to normal as the kidneys excrete alkali. When acclimatization is complete, as shown in the table, arterial oxygen saturation is only slightly below normal, but resting ventilation is increased about 20 per cent, and a compensated respiratory alkalosis exists.

Changes Due to Altitude

		Arterial Blood				
	Barometric Pressure Mm. Hg.	Oxygen Pressure Mm. Hg.	Percentage of Oxygen Sat.	Carbon Dioxide Pressure Mm. Hg.	p_H	Ventilation Ratio
Sea level.....	760	104	95	40	7.41	1.00
6,500 ft., acute....	508	73	93	39.5	7.415	1.01
6,500 ft., chronic..	508	79.5	94	33.3	7.41	1.20

Hyperventilation is the major complaint of such persons. Although relatively mild at 6,500 feet, it does lower the ventilatory reserve at rest and reduce the amount of muscular work which can be performed without respiratory discomfort. This would be of most significance in persons whose respiratory reserve was already impaired as a result of cardiac or pulmonary disease. Normal persons should not be seriously disturbed.

This type of hyperventilation is peculiar in that it is not immediately relieved by one's breathing oxygen or returning to sea level. It is thought to be due to an increased sensitivity of the respiratory center to carbon dioxide pressure.

OBSCURE FEVER IN A CHILD

To the Editor:—**A child 12 years of age, in apparently robust health, has, since the age of 4, periodically had a temperature as high as 104 F. for the past eight years. The fever persists three to six days and subsides abruptly. No other objective symptoms are present at the time. Results of physical examination and serologic tests for brucellosis are negative. What steps can be taken to establish the etiologic basis?** **M.D., Iowa.**

ANSWER.—To designate the exact nature of the disorder which is causing the child to have recurrences of fever attacks for eight years is obviously impossible. There are a number of constitutional disorders, such as tuberculosis, malaria or undulant fever, which might cause paroxysmal bouts of temperature, though as a rule the bouts, so-called, are of a more characteristic type. The diagnosis may be established by clinical and laboratory tests.

Chapters have been written on the obscure forms of fever in childhood. Many of the determining factors in the production of these febrile attacks are due to infections or inflammation in the tissues or organs of the body. During a febrile attack an attempt should be made to establish a clue by carefully examining the urine for blood, pus and organisms. The blood should be examined by not only counting cells but testing for malarial organisms and other invaders. The stools should be examined for organisms, parasites and ova of parasites. Roentgen exam-

ination may be used to investigate the viscera, bones and joints. The lungs should be examined for infiltration, cavities and acute inflammation. The heart should be examined with the stethoscope to ascertain the presence of murmurs, defective tones and rhythm; the size should be determined by roentgen examination.

In short, to determine the origin of obscure fever of the kind referred to, without any symptoms or signs indicating the site of the infection, careful and scrutinizing examinations should be made of the organs and tissues of the body and of the secretions and excretions. A hyperthyroid condition may cause fever, tremor and nervousness and may accelerate the heart action; this condition may be confirmed by a basal metabolism test. When the diagnosis is difficult, all efforts should be redoubled and continued until a satisfactory solution is obtained.

REPEATED ATTACKS OF OTITIS MEDIA

To the Editor:—**A 7 year old boy has had repeated attacks of otitis media since the age of 6 months. He has had a tonsillectomy and adenoidectomy. About six months ago his eustachian tubes were treated with roentgen rays. I have heard about radium therapy for surgically inaccessible adenoid tissue. I would appreciate information on indications, contraindications and dangers of radium therapy of this type. Penicillin does not seem to be of too much value in this case.**

M.D., Ohio.

ANSWER.—Many otologists have reported excellent results in treatment of recurrent otitis media in children by means of judicious use of the nasopharyngeal radium applicator as described by Crowe and Baylor (J. A. M. A. **112**:585 [Feb. 18] 1939), Crowe and Burnam (*Ann. Otol., Rhin. & Laryn.* **50**:15 [March] 1941), Crowe and co-workers (*Laryngoscope* **52**:790 [Oct.] 1942) and others. This treatment is indicated when lymphoid tissue is present in or in close proximity to the eustachian tube orifice which cannot be removed surgically, because of size or location, without damage to the tubal orifice or related structures. Such treatment is contraindicated when there has been previous use of roentgen rays, radium or radon type of irradiation in adequate dosage. Dangers are limited to overexposure to radiation, which should be minimal in the hands of a competent otologist with competent radiological advice. It would seem advisable that the patient in question have a complete nasopharyngeal examination, preferably by means of an electric nasopharyngoscope followed by careful surgical removal of any remnants of lymphoid tissue. If the course of roentgen treatments has not been complete, it should be completed after study of the amount of irradiation the patient has had. Any existing disease of the nasal accessory sinuses should be controlled. A complete study from the standpoint of allergy should be made, as constant recurrences could be due to this factor.

PROPHYLAXIS OF RHEUMATIC FEVER

To the Editor:—**I am interested in the reported etiologic relationship of rheumatic fever to a preceding streptococcic infection, and the association of a high antistreptolysin titer with the course of a rheumatic infection. Has any work been done on the prophylaxis or prevention of such streptococcic infections in persons subject to recurrent rheumatic fever episodes, by either peroral or injectable streptococcic vaccines, for the purpose of decreasing the incidence of streptococcic infections in such persons?**

M.D., California.

ANSWER.—Active immunization with group A hemolytic streptococci has been attempted but has not proved effective. A large scale study was made during World War II; in one instance with the type 19 hemolytic streptococcic vaccine and in another instance with the vaccine made of types 19 and 17. These vaccines were given subcutaneously. They did not protect naval recruits against throat infections with the strains of the same serologic types that were used in the vaccines (*U. S. Nav. M. Bull.* **46**:709, 1946).

SCALP INFECTION

To the Editor:—**What is the scalp infection which seems to be so prevalent in children? There are discrete vesicles or pustules usually with heavy scab formation. What is cause and treatment? It seems to be fairly self limited. Also I have been asked by women whether it is true that a person should not bathe after a sun bath.**

Anne C. Leonard, M.D., Chicago.

ANSWER.—The condition is most likely a pyogenic infection of the nature of impetigo, but, because of its prevalence at present, ringworm of the scalp should be ruled out by means of examination with a Wood light and direct microscopic examination of the hairs. If the infection is pyogenic, treatment is with antiseptic preparations, such as ammoniated mercury, or with antibiotics. The treatment of ringworm poses a more difficult problem, for it depends on various factors, such as identification of the causative organism, the age of the child, the extent of the infection and whether there are other children in the family. Water bathing does not destroy the effects of a sun bath. If the skin has been irritated by the sun light, the bath conceivably might aggravate the irritation.

PASTEURIZATION OF MILK

To the Editor:—**What differences exist between raw milk and pasteurized milk from the point of view of nutrition?**

John J. Clancy, M.D., Hartford, Conn.

ANSWER.—The pasteurization of milk is a heat treatment much less severe than that to which most foods are subjected during cooking. Reductions in nutritive value due to pasteurization have been proved in only a few instances, and these reductions do not significantly affect the over-all value of milk in the diet. Protein, lactose, butterfat, calcium, phosphorus, vitamin A and riboflavin, none of which are materially affected by pasteurization, are the main contributions of milk to the diet. Riboflavin is sensitive to light, and considerable loss occurs when milk in glass bottles is left in the sunlight for some time. However, this loss of riboflavin is not the result of pasteurization.

There is not any evidence that niacin, pantothenic acid, biotin, pyridoxine and vitamins D, E and K are significantly affected by pasteurization. Thiamine and vitamin C may be reduced by up to 20 per cent, depending on the methods and precautions observed in processing. Since milk is not a rich source of these vitamins these losses are not significant from the over-all nutritional standpoint. Articles on pasteurization by Andrews and Fuchs appeared in THE JOURNAL Sept. 11, 1948 (**138**: 128).

SYPHILIS

To the Editor:—**Please discuss the indications for retreatment of neurosyphilis, particularly dementia paralytica. In your discussion please answer the following questions: (1) What is a normal spinal fluid cell count? (2) In the case of a patient with dementia paralytica, if a spinal fluid examination done more than six months after treatment were to show a count of 1 cell or a few cells above normal and if the other spinal fluid findings had been and were changing in the direction of normality, would retreatment be indicated?**

M.D., Michigan.

ANSWER.—The management of neurosyphilis depends entirely on the observations of the spinal fluid examination. After therapy of active neurosyphilis with either penicillin or fever, the spinal fluid may show a trend toward normal values only to relapse later. It is said that relapses, however, rarely occur more than one year after therapy. If the cell count is not normal and there is no definite improvement in the other spinal fluid observations six months after treatment, further treatment is indicated. It is generally considered that any number of cells greater than 4 per cubic millimeter is abnormal, although some workers in syphilis regard any number up to 8 per cubic millimeter as normal. In the case cited, retreatment at present apparently is not indicated. Because of the slight increase in the number of cells, the spinal fluid should be examined again in three months.

CLOTTING TIME

To the Editor:—**Heparin and dicumarol® cause a prolongation of the clotting and prothrombin time, respectively; it is also stated that sulfonamide drugs and salicylates increase prothrombin time. Are there any other drugs or foods which increase either the clotting or prothrombin time?**

M.D., New York.

ANSWER.—Many drugs have been alleged to produce alterations in clotting and prothrombin time. Knowledge with respect to the effects of many of these drugs on the clotting mechanism is unsatisfactory because of conflicting reports, the different results probably being due to different methods of determination of clotting time and prothrombin time and different conditions under which the experiments were made.

Roskam (*Arch. internat. de pharmacodyn. et de thérap.* **58**:283, 1938) has reported that ergotamine, lecithin and alpha hypophamine increase the bleeding time whereas cephalin and beta-hypophamine shorten it. This has not been confirmed by other workers. Epinephrine has been reported to decrease the coagulation time, but in dogs Richardson, Hays and Walton (*J. Pharmacol. & Exper. Therap.* **73**:146, 1941) found the effect to be equivocal. Agents which produce liver damage, such as chloroform, phosphorus and *p*-toluenediamine, may increase both the clotting time and the prothrombin time.

A controversy has existed regarding the effects of the methylxanthines (theophylline and theobromine) on blood coagulation. Scherf and Schlachman reported that both the prothrombin and the coagulation time were shortened after the intravenous or oral administration of aminophylline or theophylline sodium acetate or after the oral administration of theobromine (*Am. J. M. Sc.* **212**:83, 1946). In more adequately controlled studies, Gilbert, Dey and Trump (*J. Lab. & Clin. Med.* **32**:28, 1947) did not find any changes in either prothrombin or coagulation time after administration of the methylxanthines. There are conflicting reports also with regard to the action of digitalis derivatives. Taltavull, delle Vedore and Maurelli obtained equivocal results on coagulation time after administration of digitalis, but a majority of their patients were reported to show a diminished prothrombin time (*Rev. méd. de Rosario* **36**:36, 1946). On the other hand, Supek (*Arch. internat. de pharmacodyn. et de thérap.* **77**:175, 1948) reports that in rabbits both digitoxin and strophanthin accelerated the coagulation time after intravenous injection. Strophanthin likewise decreased the prothrombin time. Pirk and Engelberg (*J. A. M. A.* **128**:1093 [Aug. 11] 1945) reported that quinine sulfate induces hypoprothrombinemia. Macht (*South. M. J.* **41**:720, 1948) reports that both penicillin and streptomycin shorten coagulation and prothrombin time. Most observers do not feel that this action of the antibiotics is of clinical importance.

No studies of the effects of foods on coagulation time appear to have been reported. Although oxalic acid occurs in some vegetables, principally spinach, it has not been conclusively demonstrated that any significant alteration in clotting time follows the ingestion of even large amounts of oxalic acid. Kohl (*Med. Klin.* **42**:535, 1947) reported that a large number of amino acids produce a reduction in clotting time in human subjects. This cannot be of any great clinical significance since enormous quantities of protein hydrolysates have been administered with only minor effects on clotting time.

Satisfactory data do not exist with regard to the effects of alcohol on clotting or prothrombin time. Foley and Wright (*Am. J. M. Sc.* **217**:136, 1949) state that excessive alcoholic intake may affect the amount of dicumarol® required to maintain the prothrombin time of patients being given this drug for cardiovascular conditions. No amplification of this statement is made.

The action of the salicylates and sulfonamide drugs on prothrombin time is of clinical significance only when large doses are administered or when there is a preexisting liver damage.

PAIN IN HERPETIC LESIONS

To the Editor:—**I desire information on the treatment of herpes involvement of the dorsal spinal nerves, with especial reference to treatment of the pain and itching manifestations.** **F. W. Lehmann, M.D., Hartford, Wis.**

ANSWER.—The results of treatment of herpes zoster in some instances may be unsatisfactory. Frequently the disease is self limited, and the pain present following the cutaneous lesions will vanish without further therapy. Those cases of herpetic involvement with considerable distressing pain not relieved by the usual methods are more often found in elderly persons and especially in women. In troublesome cases the following regimen is suggested: (1) Posterior pituitary injection twice daily for three days. (2) If no relief is obtained, give high voltage roentgen exposures to the involved or affected posterior spinal ganglion. No more than three exposures are to be given. (3) If no relief is obtained, give injections of cobra snake venom. The technic of therapy can be found in the new neurologic textbooks. (4) Paravertebral injections of procaine hydrochloride and Coley's toxin should be tried in the involved dorsal root areas. Finally, if improvement does not occur, one can suggest section of the involved posterior sensory roots (rhizotomy). Intravenous injection of sodium salicylate has been described with some success for the pain in herpetic lesions. This should be tried.

SHIGELLA ALKALESCENS AND DIARRHEA

To the Editor:—**A patient has frequent attacks of diarrhea. Blood has not been found in the stool. Shigella alkalescens has been isolated. What treatment is practical?** **A. F. Lucas, M.D., Hornell, N. Y.**

ANSWER.—Although Shigella alkalescens has been isolated from patients with dysentery, it is not considered an intestinal pathogen. It is not agglutinated by Shiga immune serum. Treatment for the eradication of this organism is not indicated, because the diarrhea cannot be attributed to Shig. alkalescens.

LOSS OF FINGER NAILS

To the Editor:—**A girl aged 18 who was examined by her physician seemed normal except for obesity. Two days later she began working at a canning factory, where she was engaged in placing hot spinach in cans. She wore rubber gloves at all times, so she had no direct contact with either the raw or cooked spinach. Two days after beginning the work, the nails on all her fingers (except the thumbs) became loosened from the nail bed and could easily be removed without causing pain. Some, but not all of the nails were surrounded by paronychial type of inflammation. The exfoliated nails did not show evidence of fungous infection. However, one of the nails is being cultured. Her toe nails are perfectly normal. Traumatic loss of the nails could be excluded because of lack of hemorrhage under the nails and lack of pain. Could some chemical reaction have occurred between synthetic rubber in the gloves and the finger nails? The skin of her fingers was normal and did not show evidence of dermatitis or trichophytic infection.** **H. J. Farrell, M.D., Milwaukee.**

ANSWER.—The information furnished suggests strong alkalies or certain cosmetics as causative agents. Suspicion may be directed to sewer drain chemicals, caustic detergents, some types of paint removers, some depilatories improperly applied, cuticle removers and some undercoating for nail polishes. Although the loss of nails appears to have been sudden, it is probable that exposure occurred considerably earlier. The work as described does not appear to be a causative factor. Previous experience indicates that these nails may return, but they may be rugose. On occasion deceit may be responsible, and household lye is one of the more readily available agents for surreptitious damage.

UNILATERAL CATARACT IN A CHILD

To the Editor:—**I have a patient aged 17 months who has wandering movements of the left eye produced by a central nuclear congenital opacity, which probably would reduce the vision to about 20/100 with a small pupil and possibly 20/60 or 20/70 with a dilated pupil, if it were not for the fact that the eye is already becoming amblyopic. The right eye is normal. Homatropine was used to dilate the pupil, but the child rubbed her eye constantly. We discontinued use of this drug for a while, but the child continued to rub the eye. Should the cataract be removed from this eye? From the literature I cannot find any specific recommendations. If it is concluded that the child will not have binocular vision as it is now, it probably will not have binocular vision even with the pupil dilated with homatropine, and will not have binocular vision after the cataract is removed.** **M.D., California.**

ANSWER.—The amblyopia and nystagmus tend to make lens opacity more noticeable, and cataract surgery for purely cosmetic purposes is in order, but not until the child is about 4 years of age. If anesthesia is given now to study the tension, it would be well also to study the right lens for any slight opacity (difficult to find without anesthesia in a young child). Unilateral cataract is certainly rare.

SYPHILIS AND PREGNANCY

To the Editor:—**A 26 year old primipara, gravida 2, has the following history: In December 1947 she contracted primary syphilis; she was treated intensively with a total of 15,300,000 units of penicillin, 0.732 Gm. of oxophenarsine hydrochloride and 1.2 Gm. of bismuth through 1948. The Kahn reaction was reported as negative June 1, 1948, by one laboratory and positive by another one. A Kahn reaction of the spinal fluid was negative. No further treatment was deemed necessary except local treatment for a persistent erosion of the cervix. The Kahn test of the blood, made at intervals, elicited negative reactions except one, on March 11, 1949, which was positive. Results of tests made since then were negative. What treatment, if any, is indicated during this period of gestation?** **M.D., North Carolina.**

ANSWER.—It is probably unnecessary to administer more antisyphilitic treatment to this woman as it is in the vast majority of women who have had adequate antisyphilitic therapy prior to pregnancy. When one is in doubt about the status of a case, however, it is well to administer penicillin, 300,000 units daily for ten days.

BRONCHITIS

To the Editor:—**Is bronchitis hereditary?** **Edward J. Grass, M.D., Grand Rapids, Mich.**

ANSWER.—Bronchitis is not inherited but is contracted after birth. Bronchitis in children usually is due to infection or allergy. Acute bronchitis may become subacute and later chronic. In such instances there is often a focus of infection in the mouth, paranasal sinuses or the terminal bronchi. Pulmonary congestion as occurs in mitral stenosis may result in symptoms of chronic bronchitis. Pressure on the larger bronchi caused by enlarged lymph nodes and aspirated foreign bodies result in symptoms often attributed to chronic bronchitis. Congenital bronchiectasis has been described and may be mistaken for chronic bronchitis. However, there is no evidence that bronchiectasis is inherited.